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THE

CLINICAL JOURNAL

CLINICAL RECORD, CLINICAL NEWS, CLINICAL GAZETTE, CLINICAL REPORTER, CLINICAL CHRONICLE AND CLINICAL REVIEW

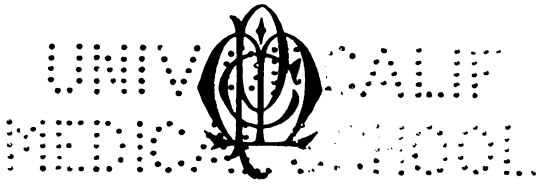
A WEEKLY RECORD OF CLINICAL MEDICINE AND SURGERY, WITH THEIR SPECIAL BRANCHES

IN TWO VOLUMES ANNUALLY

VOL. XXXVI.

APRIL 13—OCTOBER 5, 1910.

EIGHTEENTH YEAR.



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LIST OF CONTRIBUTORS.

PAGE

ANDREWS, HENRY RUSSELL, M.D., B.S., M.R.C.P. Some Practical Points in the Management of Difficult Labour 251

BATTLE, W. H., F.R.C.S. In the Wards of St. Thomas's Hospital 312
BOSANQUET, W. CECIL, M.D., F.R.C.P. Some Considerations on Pleurisy 213
BUNCH, J. L., M.D., D.Sc., M.R.C.P. Skin Rashes and their Treatment... 257
BUZZARD, E. FARQUHAR, M.D., F.R.C.P. The Early Diagnosis of Subacute Combined Degeneration of the Spinal Cord 374

CAMPBELL, HARRY, M.D., F.R.C.P. Medicine on the Continent 53
CAUTLEY, EDMUND, M.D., F.R.C.P. Simple Infantile Anæmia 325
CORNER, EDRED M., B.Sc., M.B., M.C., F.R.C.S. Treatment in Septic Conditions 151
CUMSTON, CHARLES GREENE, M.D. The Diagnosis and Differential Diagnosis of the Pathological Processes causing Enlargement of the Kidney ... 377
CURTIS, HENRY, B.S., F.R.C.S. Interesting Cases of Abdominal Surgery, including some Rare Forms of Intestinal Obstruction 22, 42

DUNN, PERCY, F.R.C.S. So-called Second Sight—its Features, Varieties, and Causes 122

EDWARDS, F. SWINFORD, F.R.C.S. Lecture on Abscess and Fistula 401
EVE, FREDERIC, F.R.C.S. Three Rare Tumours of the Base of the Tongue (two of them Endotheliomata), with Remarks on the Surgery of Malignant Disease of the Tongue 113

GRÜNBAUM, OTTO, M.D., F.R.C.P. Clinical Lecture on Arthritis Deformans 289
GUTHRIE, LEONARD G., M.D., F.R.C.P. On Acute Bulbar Paralysis (Polio-encephalitis Inferior) in a girl, æt. 13 years... 385

HADLEY, W. J., M.D., F.R.C.P., F.R.C.S. Clinical Lecture on Diseases of the Colon 337
HAWKINS, HERBERT P., M.D., F.R.C.P. Treatment of Colitis 33
HERRINGHAM, W. P., M.D., F.R.C.P. Clinical Lecture on Chronic Ascites with Cirrhosis of the Liver 65
HERSCHELL, GEORGE, M.D. The Non-Surgical Treatment of Ulcer of the Duodenum 273
HERTZ, ARTHUR F., M.A., M.D., F.R.C.P. Clinical Lecture on Asthma 198
" " Clinical Lecture on Muco-membranous Colitis ... 263
HOLMES, GORDON, M.D. Two Clinical Lectures on Hemiplegia and its Treatment ... 129, 156
HUTCHISON, ROBERT, M.D., F.R.C.P. Lecture on Hysteria in Childhood 305

LANE, W. ARBUTHNOT, M.S. Iliac Constriction in Intestinal Stasis 16
LETT, HUGH, M.B., F.R.C.S. Clinical Lecture on Retention of Urine 281
LEWERS, ARTHUR H. N., M.D., F.R.C.P. Case of Pan-hysterectomy for Carcinoma of the Body of the Uterus, in which an Infiltrated Portion of the Bladder was Removed 6

		PAGE
LOCKWOOD, C. B., F.R.C.S.	Clinical Lecture on some Fundamental Truths in Surgery	1
,, ,,	Inguinal Ruptures which are Difficult to Manage, and their Treatment	177
,, ,,	Lecture on Femoral Ruptures and their Management	209
,, ,,	Lecture on Radical Cure of Femoral Hernia	321
MORTIMER, J. D., M.B., F.R.C.S.	On Two Common Errors in the Administration of Anæsthetics	63
MURRAY, GEORGE R., M.A., M.D., D.C.L., F.R.C.P.	Clinical Lecture on Fibrositis of the Abdominal Muscles	49
ORMEROD, J. A., M.D., F.R.C.P.	Clinical Lecture on a Case of Cerebral Tumour	343
ORMOND, ARTHUR, F.R.C.S.	Sympathetic Ophthalmia	70
PERNET, GEORGE, M.D.	Lecture on Lupus Erythematosus	221
POYSER, A. V., M.B., B.C.	The Hyperæmic Treatment of Inflammation	139
RANKIN, GUTHRIE, M.D., F.R.C.P.	Migraine	247
RIVIERE, CLIVE, M.D., F.R.C.P.	A Clinical Lecture on some Cases of Tubercular Disease ...	302
SANDWITH, F. M., M.D., F.R.C.P.	Smallpox and its Early History	294
,, ,,	The Life and Work of Edward Jenner	328
,, ,,	The Practice of Inoculation of Smallpox	317
,, ,,	Value of Vaccination	346
,, ,,	Two Historical Cases of Cancer and the Treatment of the Disease	8
SHAW, H. BATTY, M.D., F.R.C.P.	Clinical Lecture on Asthma	86
SPRIGGS, E. I., M.D., F.R.C.P.	The Treatment of Indigestion	173
STEWART, PURVES, M.D., F.R.C.P.	Clinical Demonstration at the Polyclinic	353
TAYLOR, FRANK E., M.A., M.D., M.Sc., F.R.C.S., M.R.C.P.	Case of Myomectomy during Pregnancy	47
THOMPSON, A. HUGH, M.A., M.D., B.C.	On Some Affections of the Eye-muscles	29
,, ,, ,,	On Some Pitfalls in the Diagnosis of Common Diseases of the Eye	286
THURSFIELD, J. HUGH, M.D., F.R.C.P.	Lecture on Meningococcal Meningitis	407
WALL, CECIL, M.D., F.R.C.P.	Foreign Bodies in the Air-passages	161
WALLIS, F. C., F.R.C.S.	Clinical Lecture on Sigmoiditis and Proctitis	241
,, ,,	Clinical Lecture on some Cases of Tubercular Disease	81
,, ,,	A Demonstration at Charing Cross Hospital on Rectal Fistula ...	369
WARING, H. J., M.S., F.R.C.S.	Clinical Lecture on a Case of Endosteal Sarcoma of the Tibia	17
,, ,,	Two Cases of Carcinoma of the Rectum Treated by the Abdomino-perinæal Method	193
WATSON, C. GORDON, F.R.C.S.	Innocent Tumours of the Rectum	389
WEST, SAMUEL, M.D., F.R.C.P.	Three Clinical Lectures on the Heart 97, 118,	137
WHITE, W. HALE, M.D., F.R.C.P.	Clinical Lecture on a Case of Syphilis	145
WILLIAMS, LEONARD, M.D., M.R.C.P.	Pituitary Extract and Some of its Effects	92
WILLIAMS, TOM A., M.B., C.M.	Aphasia and other Disorders of Speech	268

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No. 911. WEDNESDAY, APRIL 13, 1910. Vol. XXXVI. No. 1

CONTENTS.

PAGE

*A Clinical Lecture on Some Fundamental Truths in Surgery. By C. B. LOCKWOOD, F.R.C.S. 1

*A Case of Pan-hysterectomy for Carcinoma of the Body of the Uterus, in which an Infiltrated Portion of the Bladder was Removed. By ARTHUR H. N. LEWERS, M.D., F.R.C.P. 6

*Two Historical Cases of Cancer and the Treatment of the Disease. By F. M. SANDWITH, M.D., F.R.C.P. ... 8

Iliac Constriction. By ARBUTHNOT LANE 16

** Specially reported for the Clinical Journal. Revised by the Author.*

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A CLINICAL LECTURE

ON

SOME FUNDAMENTAL TRUTHS IN SURGERY.

Delivered at St. Bartholomew's Hospital.

By C. B. LOCKWOOD, F.R.C.S.,
Surgeon to the Hospital.

GENTLEMEN,—I propose to-day to lecture upon a subject which is controversial and obviously open to differences of opinion. But before the end you will agree with me that it is one to which we ought all of us give our most earnest consideration. I propose to discuss three propositions. First, that it is our duty as medical men to save the lives of our patients; second, that it is our duty to prolong the lives of our patients; and third, that it is our duty to relieve pain and distressing symptoms. Let us begin with the first proposition, which says that it is our duty to save the patient's life. I often wonder whether the public are not less solicitous on this point than we are ourselves. It is a matter of common observation that when a man's wife or child is desperately ill he runs off to the first medical man and places those lives unreservedly in his hands. But now see what happens if by chance the man has quarrelled with another and struck him, and there is a fatal ending. Now all the machinery of the law is put into motion. I suppose two policemen will take him off. After a while a solicitor prepares his case, briefs a barrister, then he is taken into court, there is a judge, there is a jury, there are counsel and solicitors on the opposite side. In fact a huge organisation is put into motion, and no expense is spared. Why this difference? A cynical friend of mine suggested that the first was a natural death, whilst the second was a shameful one. However, there is a much more obvious reason for

this difference. The peril of the wife or child has to be met instantly—at the moment. Hours, perhaps minutes, are of great importance. But the machinery of the law takes a long time to be put in motion, and by the time it reached its end I suppose the patient might be dead. Now see what has to happen. The medical man who is called in to this grave case has to act there and then upon his own responsibility. He has to act as solicitor, as counsel, as judge, and as jury, and perhaps has to put the verdict into effect—obviously a very grave responsibility for any human being, and one which no one would lightly undertake unless he was fitted for it by training and education. Now, in discussing this first proposition, that it is our duty to save the life of the patient, I shall illustrate what I am about to say by certain examples. But I need hardly warn you against being misled by those who choose particular instances to prove their arguments. If you are allowed to choose particular instances you can prove anything. To prove cases by the choice of particular instances the audience must be ignorant and stupid, which you are not. The instances which I am about to give are mere illustrations. You may have heard the story of the philosopher who was taken to see the temple of Neptune. Around the walls were the pictures of those who had made dangerous voyages in ships like those of St. Paul. And naturally they were glad whenever they touched land again, and their votive offerings were hung in the temple of Neptune. The philosopher was asked to gaze on the pictures and acknowledge the goodness of the sea god, the preserver of the givers. But he said: "Now show me the pictures of those who went to sea and did not come back?" So when wonderful cures are told, ask about those who were not cured. When it comes to a question of saving a person's life, the decision is, as a rule, obvious and clear. Over and over again you have opportunities of seeing this. But I am now going to bring before you a hard case which will convey to your minds the impression that the decision is not always quite so easy as you think, and that unless the mind of the medical man be guided by fundamental truths and first principles he might easily falter in his decision. Here is an instance —I am going to alter matters a little—of an old man, one who had almost reached the allotted span of three score years and ten, and who in reasonable probability might not have many years to live. He not only is old and well off, but in addition he is an imbecile, and he has been for years in bed, at considerable expense to himself, and of trouble and anxiety to his relatives: two nurses are constantly in attendance, and now at last he gets an attack of obstruction which, if unrelieved, must end inevitably in his death. What are you going to do for that man? He is old, he is imbecile, and he has a fatal disease. You will be asked, if you operate will he get better? You will be asked, if you operate will he be cured? It will be suggested to your mind what is the good of operating on him when he is so old and when he is imbecile? I venture to think that under such circumstances as that it is our duty as medical men to put everything aside and say that it is our duty to save the life of this patient. Take the argument of age; what has that got to do with it? The other day in Lucas Ward there was a woman, æt. 76 years, who two years ago was operated upon for strangulation of an obturator hernia on the right side. The other day she was operated upon for strangulated obturator hernia on the left side and recovered. What pleasure she is likely to derive from the remainder of her existence is nothing to do with me. I do not know. But plainly our duty was not to leave her alone because she was old, but to endeavour to save her life. An old lady, in her eighty-fourth year, had an acute appendix abscess opened and her appendix removed, and then lived happily for three years. Next, our patient was an imbecile. What has that to do with it? If you consider the attitude of the State towards imbecility and lunacy, you will find that it decrees that the greatest care shall be taken of them. Furthermore, the people in lunatic asylums are not allowed to die of fatal diseases; they are operated upon, and if possible, saved. You see occasionally advocated—I saw it recently advocated—that the State should at once deal with the mentally afflicted and get rid of them. I suppose the ideal lunatic asylum of that person would consist of a receiving room, a lethal chamber, and a crematorium. But just imagine if once you began to condemn human beings to death because of their mental condition, where you would end. Surely I cannot help suspecting you will find people who might refrain from operating because they did not like the person's nationality, or perhaps even his theology, or his political creed. Surely we as

medical men have nothing to do with these things. It is our bounden duty, when brought into contact with a dying person, to try if possible to save his life, quite irrespective of his mental condition. There are a great many other considerations which occur when you are brought into contact with a dying or moribund person. You may allow your mind to be influenced by all sorts of surrounding circumstances. You may say: "It will not be good for my reputation to have a fatal result placed to my discredit." Such considerations should have no weight whatever in your minds. You may say, "This patient is so desperately ill that he cannot possibly recover." I will now give you a few instances of patients that I have operated upon, and who at the time seemed to have hardly any chance of recovery, but who yet got well. Of course I am only taking you to the temple of Neptune. How many did not recover? I can recall a patient of Dr. Herbert Newton's who I thought was moribund. There was no question about the reason for his perilous condition. Pus was oozing from his right ear; he was paralysed down all the left side of the body. His breathing was stertorous. I need not say that he was absolutely unconscious, and should have thought it was practically hopeless to do anything to save him. And I had another reason for thinking that he was a hopeless case. A cerebral abscess in the temporo-sphenoidal lobe is not a hopeless condition at all. Such an abscess after opening and draining not infrequently ends in recovery. But this patient had a symptom which led me to think that in addition to the abscess he might have basilar meningitis, because he had paralysis of the fifth nerve on the left side, the left conjunctiva was insensitive, and we thought he had some facial paralysis on that side. He was operated upon and recovered in spite of all that. Some years afterwards this man came to the surgery, and the only relic of his abscess which he had had in his temporo-sphenoidal lobe was occasional epilepsy, which was quelled by doses of bromide of strontium. So that if I had not gone back on fundamentals, and determined that apart from every consideration it was my duty to attempt to save that man's life, I do not think he would have been with us now. I will tell you of another case. A girl had an abscess after middle-ear disease. She was taken to the theatre and a portion of her occipital bone was removed. The dura was opened and the cerebellum bulged. She ceased breathing, and artificial respiration was begun, and continued for four hours, after which she died. But this is the point: that I ought, whilst the artificial respiration was going on, to have explored the cerebellum, because she had an abscess there which might have been opened. I often think of a case which it is difficult to tell to you. Eminent men were in consultation upon a youth who was supposed to have middle-ear disease. He had had a blow upon his head. He was in much the same condition as the man of whom I have just spoken. It was thought by those in attendance that he had a temporo-sphenoidal abscess, and they advised that it should be opened. Further opinions were sought for, and great doubt was cast upon this diagnosis; in fact a positive diagnosis of septic meningitis was given. If I had been the surgeon who gave the first opinion I should have considered it my duty to try to save the life of the patient irrespective of anything. But the surgeon who was in attendance declined to proceed, and another surgeon opened the skull and demonstrated the septic meningitis.

I have told you of successful cases so far, but I might spend the rest of the time in telling you of unsuccessful ones. How far are you to allow yourselves to be deterred in your efforts to save the life of the patient by a series of unsuccessful cases? I can only say if you are to be deterred by unsuccessful cases you will not achieve much. I was one of the earliest to operate with avowed intention of trying to cure diffuse septic peritonitis. The number of patients who did not get better was very great, but at last a case did get better, and now you are aware that cases of diffuse septic peritonitis get better in a larger proportion than formerly. I could recall the attempts to save those with perforating ulcer of the duodenum. I remember publishing some of the first unsuccessful cases. And perforating ulcer of the duodenum was operated upon unsuccessfully over and over again, until my friend Mr. Eve published, if I remember rightly, the first success. And now perforating ulcer of the duodenum is often operated upon successfully. If you think it is right to operate, do not be deterred by a series of unsuccessful cases. I might give other instances, but I merely wish to instil the thought into your minds.

I shall not say anything about the risks which you are to take during the course of an operation in endeavouring to save a patient's life, for the very simple reason that we are not concerned with the risks which are to be taken during the course of an operation; we are here concerned with the preliminary act of judgment, which will determine you that it is right to take those risks. Another train of circumstances which may affect your judgment, and which I propose to mention to you, are cases such as those in which legal proceedings may follow your attempts to save the patient's life. For instance, there is a fracas in a public house, a revolver is produced, and the potman is shot in the abdomen. It is clear that if he is left alone he is likely to die. Operated upon, you may save his life. In coming to this decision your mind must naturally dwell upon what may follow. The patient may die. You perhaps can imagine the cross-examination to be undergone by you at the trial of the person who fired the shot. You will be asked if the bullet had been left might not the patient have recovered? You will be asked how long after your operation the patient died. There was a notorious case some years ago which I only know by hearsay, in which a surgeon operated upon a policeman, who had been shot, and I suppose that the surgeon did not come out of the ordeal of cross-examination well because the person who fired the shot got off. But I am told by those in a position to know, that for years afterwards the little boys shouted after this surgeon when he drove about the town, "Who killed the peeler?" That cannot have done the poor surgeon's reputation any good. But such considerations as that should have no weight whatever in your minds. Your business is to put aside everything and proceed there and then to do the best you can to save the life of the patient.

Now I come to the second proposition, that it is our duty to prolong the patient's life. To take a common instance we see the attempt made to prolong the patient's life in cases of malignant disease of the rectum. In those cases an inguinal colotomy is performed. The patient is not cured, but his life is usually prolonged by that operation. Mind this, if you watch the subsequent history of some of these cases of inguinal colotomy you will find that it is miserable. Females in particular feel acutely the horrors of an inguinal colotomy. I do not know anything more distressing and disgusting than an ill-performed inguinal colotomy. But your business, I venture to think, is not to consider what may or may not be the future of that patient, but to endeavour to prolong her life. Remember many after inguinal colotomy live in comfort for years, and I have often seen people who have had inguinal colotomy performed who were perfectly contented with it, and show off the colotomy with some degree of interest and almost of pride. Further considerations arise in some operations, such, for example, as those for the removal of cerebral tumours. A person who removes a large cerebral tumour is aware that the patients may be left with some amount of paralysis, that their mental condition may be unsatisfactory, that they may have epileptic fits. I maintain that they are right in performing these operations to prolong the patient's life, and that it is not for the surgeon to form an opinion as to what the patient will think of the condition in which he leaves him. It may be unsatisfactory. But on the other hand it may be satisfactory. Sometimes efforts are made to prolong life in very desperate cases. I recollect a prison warder who had just become entitled to his pension, who was sent up with the most extensive carcinoma of the whole tongue, and of the lymphatic glands of the neck. His seemed a hopeless case for operation. He was shown to my colleagues in consultation, and they urged that some effort should be made to prolong his life. They knew of course that if no effort was made his days would be short and miserable. That man had a sweeping operation performed, and I know that a year afterwards he was well. Now, if I had been asked, and trusted my memory, I should have said that he was well five years afterwards, but I advise you not to place too much credence upon these wonderful efforts of memory, especially when that which is remembered is something pleasant. I wrote to my friend Dr. Griffith, who knew all about this patient, and he told me that the man died eighteen months after the operation. So at any rate he had a complete year of comfort, and I maintain that the effort to prolong life in that particular instance was fully justified. I can recall another desperate case, which is even better. A lady came up from the Midlands where she had had a totally inadequate operation performed for carcinoma of the left

breast. She had a large ulcerating carcinoma of the left breast and carcinoma of the axillary glands. Altogether it was a desperate state of affairs. In addition there was a very strong suspicion of the presence of growth in the glands above the clavicle. She was seen in consultation by Mr. Willett, and he, too, thought she had carcinoma in the glands above the clavicle, but that an effort should be made to give relief. At any rate we thought at that time to prolong her life but not to cure her. Fortunately for her the nodule which was felt above the clavicle when she was first examined was found not to exist; it was the omo-hyoid, which contracted and swelled up when touched. A sweeping operation was performed for removal of the breast and the pectorals and glands. A year afterwards, under gas, a small nodule of carcinoma was removed from the scar. Four years afterwards a nodule of carcinoma appeared beneath the clavicle and adherent to the first intercostal space close to the cartilage of the first rib. With great trouble that was removed. The axillary vein had to be tied at its junction with the subclavian. A nodule of cancer was dissected off the axillary artery. This patient died of an abdominal tumour thirteen years after the first attempt.

Do not always act as if your diagnosis were infallible. An old lady in Lucas Ward was supposed by everyone to have a malignant tumour in Douglas's pouch. She had a hard abdominal tumour with all the characteristics of malignant disease, and it was diagnosed to be such by most competent people. However, when it was looked at, this mass of malignant disease was found to be a very rare and curious condition, namely, a small ovarian cyst which had become twisted on its pedicle and fallen into the pelvis, and of course become inflamed and adherent. The operation was comparatively easy and the patient was cured.

Now I come to the last proposition: That it is your duty as medical men to relieve pain and distressing symptoms. You have to make up your mind what you are going to do about the administration of morphia to people who are in pain. It is not our business to shorten the patient's days. If it is clear to your mind that the administration of morphia for the relief of pain will shorten your patient's life, I do not think you ought to administer it. For instance, you are called in to see a patient in the last stages of some dreadful abdominal crisis, such as acute septic peritonitis, and you know perfectly well that a dose of morphia will produce peace and calm but it will accelerate that patient's end. I do not think that I myself should care to incur that responsibility. But nevertheless it is a point about which you have to be perfectly clear in your own minds. On other occasions when the copious administration of morphia does not jeopardise recovery, I have unhesitatingly advocated that it should be given. For instance, a patient was operated upon for carcinoma of the uterus, and operated upon again and again. And at last the disease spread to the pelvic organs and to the sacral plexus, and the agony was unendurable. Surely in such a case as that it would be improper and wrong not to do all one could, by morphia or other means, to relieve that person's anguish. I remember another dreadful case, of a patient who was supposed to have adenoma of the left breast. It was removed, and instead of being examined at the time was examined later on, and found to be carcinoma. A year later a tumour grew in the right breast. A sweeping operation was done but it was an innocent adenoma. Then the right shoulder became painful and stiff. She had been told that if she waited it would become ankylosed and the pain cease. Massage and electricity rapidly improved the shoulder, but there were a number of enlarged carcinomatous glands in the left axilla. These were removed by a sweeping operation. Nothing further happened until a year after, when rheumatic pains in the left hip were reported, but nothing could be discovered. One day the left femur broke. The agony was horrible —one continuous shriek. How wrong in such a case as that to withhold every possible means of alleviating anguish. However, you have to make up your own minds on these matters. There is another point about the relief of pain. I once instigated an operation for the relief of pain. I am not referring to neurotomies or neurectomies, but to another class of case. I was asked to see a man who was obviously in the very last stages of septic peritonitis. I should not have thought there was any hope of his recovery. Perhaps he had a faint chance, but that man was enduring the agonies of the damned. I could not open his abdomen myself to let out the pus and drain and stop the pain, but I asked Mr. Harmer to do it for me and

it was done. No further pain and no further anguish or vomiting. But the unfortunate man died. I can recall another case of the same kind, where, in the very last stages of septic peritonitis, due, I suspect, to a perforated appendix, the patient was enduring the most appalling agony. He would have done anything to be quit of his pain and of his life. I did not think it possible he could survive any operation. But I said this: Provided the anæsthetist could give him an anæsthetic for, say, ten minutes, I could evacuate the pus and relieve the pain. I said to this unfortunate gentleman: "If you will take a little anæsthetic I can do an operation which will relieve you of your pain." The abdomen was opened and an enormous quantity of pus evacuated. He had a peaceful ending. There may be other instances which do not occur to my mind, but I have operated more than once to relieve vomiting, I have performed gastro-enterostomy on people at the last gasp because they were dying of vomiting, and I submit it is kind and proper to do gastro-enterostomy under such circumstances as that, if the only prospect you can hold out is euthanasia. Another operation which I sometimes perform reluctantly is gastrostomy, not in the hope of curing the carcinomatous stricture of the œsophagus, but can you imagine to yourselves what it must be to die of thirst? I think it is one of the most dreadful endings that a human being can possibly come to, and on more than one occasion I have said to these unfortunate people that if they were prepared to take an anæsthetic and have an operation performed I would promise to cure them of their thirst, which of course the operation does.

I have placed before you, gentlemen, a very wide and, I think, a very important topic, and it is one about which I trust you will begin to think deeply. It is clear that you must come to convictions of your own on these three propositions, because if you have not convictions of your own I am afraid you will not act properly or wisely when you are confronted with these dreadful emergencies.

April 11th, 1910.

DR. DOYEN will commence on April 18th a course of fifteen lectures on "Surgical Anatomy" at the Paris Faculty of Medicine, open to all students and doctors.

A CASE OF PAN-HYSTERECTOMY FOR CARCINOMA OF THE BODY OF THE UTERUS, IN WHICH AN INFILTRATED PORTION OF THE BLADDER WAS REMOVED.

By ARTHUR H. N. LEWERS, M.D., F.R.C.P.,
Senior Obstetric Physician to the London Hospital.

E. H—, a single woman, aged 60 years, was admitted into the London Hospital, under my care, on June 3rd, 1909. She complained that for about a year she had been losing blood, sometimes every day, sometimes with an interval of a week. For about the same time she had complained of pain in the left hip and down the left leg. The symptoms came on after a fall. For about a month prior to admission there had been increased frequency of micturition. The menopause occurred at about the age of 53. Since that time she had "seen nothing" till the bleeding began again as mentioned above. Prior to the menopause menstruation had always been regular and normal.

Nothing abnormal was found on abdominal examination. On vaginal examination the cervix appeared healthy. Bimanually the body of the uterus was felt to be distinctly enlarged, and its outline suggested that there might probably be a fibroid present. The uterus was freely movable. The urine was normal.

The diagnosis arrived at was cancer of the body of the uterus, with probably one or more fibroids present as well.

On opening the abdomen, June 25th, 1909, several adhesions between the large intestine and the back of the uterus and pelvic floor were found. These were separated, several bleeding points on the intestine requiring ligature. A puckered condition of the peritoneum was seen in the position of the utero-vesical pouch. An expanding retractor was placed in the lower angle of the wound to obtain as good a view of the pelvic contents as possible. A volsella was now applied to the fundus of the uterus. The uterine tissue began to tear at once, causing a slight leakage of obviously malignant growth from the tear. A nodule the size of a walnut could be felt on the anterior surface of the body of the uterus, to which the bladder and its

peritoneal coat seemed firmly adherent. At this stage the condition of the lumbar glands was examined with a view to determine if it was worth while proceeding with the hysterectomy. As no enlargement of the glands could be felt, the operation was continued. The upper parts of both broad ligaments were tied off in the usual way, so as to allow of the uterine appendages on both sides being removed with the uterus. The next step—the separation of the bladder—was then proceeded with. On attempting to separate the peritoneum and bladder from the region of the nodule mentioned, on the anterior wall of the body of the uterus, a very vascular area of the uterine tissue in that situation was exposed, and its appearance strongly suggested that the malignant growth had extended to the fundus of the bladder at that level. The corresponding area on the bladder-wall was hard, and seemed almost certainly invaded by the uterine growth. It was, however, found to be quite at the upper part of the bladder, and the hard area was distinctly circumscribed, so that it seemed quite possible to remove the suspected portion of the bladder-wall. For the moment the removal of the uterus was proceeded with. The uterine arteries were tied, the vagina opened anteriorly first, then the posterior wall was divided, and finally the lateral attachments. Various bleeding points on the vaginal walls were ligatured. It was found that there had been some escape of carcinomatous tissue into the vagina. This was removed as completely as possible. It remained to deal with the nodule on the fundus of the bladder. The nodule was held up, and the bladder wall incised all round it with scissors at a distance of about $\frac{3}{8}$ of an inch from the hard periphery. When this had been done, there was left an opening into the bladder the size of a five-shilling piece. The opening was closed first by a continuous catgut suture, just missing the mucous membrane, and then by a row of interrupted sutures, also of catgut, over the continuous suture. A gauze drain was passed down into the vagina, and the peritoneum closed over it. The abdominal wound was closed in the usual way. A soft catheter was left in the bladder, and fixed by a stitch to the vulvar skin.

The patient made a good recovery, and left the hospital on July 23rd. The urine remained acid throughout. Up to the end of the first week it contained a little blood, but not afterwards.

Description of the specimen.—The parts removed at the operation consist of the uterus and its appendages, and of the portion of bladder invaded by the growth. This portion forms a small ovoid mass the size of a walnut.

The uterus measures about $3\frac{3}{4}$ in. in length. The whole of the endometrium is the seat of a malignant growth, which has in every direction deeply invaded the uterine wall, in places up to the peritoneal coat. In one place—that corresponding to the place where the bladder was adherent—the growth extends up to the free surface. The cervix is healthy. There is a small fibroid—the size of a large walnut—seen on the right side of the uterus, growing out into the broad ligament below the attachment of the right uterine appendages. Microscopically the malignant growth is a glandular carcinoma.

Sections for the microscope were taken—(1) Through the nodule in the bladder-wall, where the anterior surface of the uterus was adherent to the bladder. (2) Through the infiltrated anterior wall of the uterus.

Dr. R. D. Maxwell, Obstetric Registrar at the London Hospital, reports as follows: "*Section No.* 1 shows the subserosa to be thick and fibrous; in it are a few islands of shrunken cells, similar in appearance to those in the section of the uterine growth. *Section No.* 2 shows the growth in the endometrium to be composed of columnar cells in tubular arrangement."

Remarks.—I have never before met with a case of carcinoma of the body of the uterus in which the growth has spread to the bladder in so limited a manner as to make it reasonable to suppose that excision of a portion of the bladder-wall would completely remove the infiltrated tissue. In the case here recorded the invasion of the bladder-wall was quite circumscribed, and after the nodule there had been excised, the cut edges of the bladder surrounding the gap so produced appeared quite healthy. This impression was confirmed by the fact that the wound in the bladder healed soundly. A consideration of the circumstances of the case as described above also makes very clear the superiority of abdominal hysterectomy over vaginal hysterectomy in affording an opportunity for dealing with possible complications, many of which, such as the invasion of the bladder in this case, cannot be recognised beforehand.

A point of minor interest is the presence of a

fibroid co-existing with carcinoma of the body of the uterus. In my experience of these cases, which has been considerable, this association of fibroids with carcinoma of the body is very common—so much so that I should be inclined to say it occurs in a majority of cases.

The presence of a wound in the bladder—intentional, of course, in the case just recorded—leads me to say a word about the suturing of such wounds, which are occasionally met with as unintentional complications in the course of gynæcological laparotomies, especially in certain cases of hysterectomy for large uterine fibroids, and in cases of very large broad ligament cysts.

I have performed considerably more that 1000 laparotomies, and my experience of unintentional wounds of the bladder of this kind, so far as I can remember, has consisted of three cases. One was a case of large retro-peritoneal cervical fibroid, where the highest point of the tumour reached the epigastrium. In this case the bladder was very much thinned, being dragged up as high as the umbilicus, and tightly stretched over the front of the tumour. In stripping down the bladder a hole was torn in it some two inches long. This was sutured with a continuous suture of fine silk and a second row of interrupted silk sutures over the continuous one. The wound healed uneventfully, and gave no further trouble. Another case was also one of abdominal hysterectomy for fibroids, where the same accident occurred, and where similar treatment was quite satisfactory. A third case was one of retro-peritoneal broad ligament cyst, the size of the uterus at term. In this case the bladder was displaced to the right, very much thinned and drawn up, and spread over the *right* side of the cyst. In incising what was thought to be peritoneum on the right side, the bladder was so extensively opened that it lay in two halves, united behind like an open book. In this case a continuous catgut suture, avoiding, of course, the mucous membrane, was used first, and over this a series of interrupted catgut sutures. A drainage-tube and gauze drain were left in the lower angle of the abdominal wound in this instance, in case of leakage; but nothing of the kind occurred, and the wound in the bladder gave no further trouble. The fourth instance in my practice was the intentional wound in the bladder described in the case forming the subject of the paper. As regards these four cases, in two of them the bladder was sutured with fine silk, and in two with catgut. In two of them a soft catheter was left in the bladder for some days; and in the other two a catheter was passed at frequent intervals. Silk and catgut seemed to do equally well as a suture material, and the cases where a catheter was passed frequently after the operation did just as well as those in which a catheter was left in.

April 11th, 1910.

TWO HISTORICAL CASES OF CANCER AND THE TREATMENT OF THE DISEASE.*

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LECTURE III.

It is not possible to describe to you the early symptoms in all the different organs in which cancer occurs, or what first induces the patient to consult the doctor. Let us, however, consider an example, say a case of cancer occurring in the tongue. This is not unusual among men of forty-five or older who have indulged in excessive smoking, especially of foul pipes. Another cause may be the jagged point of a tooth which has rubbed the tongue for some time, and has caused first a sore at the side of the tongue and finally a chronic swelling or ulcer. Sometimes the first sign of disease observed may be a crack or fissure, or a warty growth. In whatever way it starts a hard new growth is soon noticed, which may or may not be painful, which ulcerates and bleeds freely when touched. All the movements of the tongue become painful and difficult, swallowing and speech are greatly affected, the pain extends to the ear and throat, and sleep becomes well nigh impossible. The glands of the neck are involved, swell and burst outwardly, the lower jaw is sometimes invaded. The difficulty in taking sufficient nourishment, the extreme suffering and the exhaustion rarely allow the patient to live for more than a year after the first symptoms are noticed.

Such a case is recognised by the surgeon as one of cancer when there is early enlargement of the glands under the jaw, profuse salivation and difficulty in moving the tongue. But it is all-important for the patient's sake that diagnosis should be made early, and therefore, when there is a doubtful lump, it is better to cut off a small portion of the growth and have it examined under the microscope by a competent authority. For such an operation local anæsthesia may be induced by cocaine.

The face is more liable to cancerous infection than any other part of the skin, perhaps because it is more exposed.

* Three Lectures delivered at Gresham College.

In order to give you some idea of the course of this illness I will tell you what is known of one or two well-known patients who have died from it, and whose symptoms are, on account of their personality, of universal interest.

I have already mentioned the fact that the father of Napoleon I died of cancer of the stomach, and when the great Emperor himself began to suffer from severe and increasing trouble in the stomach he at once concluded that he had the same disease. Some doubt has been thrown upon the diagnosis of late years, and as the medical treatment he received on the island of St. Helena was, at least according to our modern notions, very inadequate, it will never be quite satisfactorily settled. But judging by the length of the illness, which lasted for nearly four years, and also by some of the symptoms, we may conclude that he had at first an ulcer of the stomach, in which a cancerous growth afterwards developed, causing his death.

Dr. Warden, surgeon on board the "Northumberland," which conveyed Napoleon to St. Helena in 1815, was on very friendly terms with Napoleon, and he tells us how the ex-Emperor rallied him on his habit of blood-letting for all diseases, which was, of course, the method of treatment then most commonly used. He says that Napoleon was an indifferent sleeper, and that his pulse never exceeded 62. "Of his own state of health" his letters continue, "he has good reason to boast, and when it is considered to how many various climates he has exposed himself, and what a succession of toil he has undergone during the last twenty-five years, the state of health he has enjoyed and still enjoys is astonishing. He declares that he has been but twice, throughout his life, in such a state as to demand medical aid. He took a dose of physic for the first complaint, and the second, being a pulmonary affection, on his return from Egypt, required a blister."

But soon after his arrival on the island Napoleon began to be troubled with digestive difficulties. He was a moderate eater, and, considering the times he lived in, he was a remarkably moderate drinker, for those were hard drinking days, but he accounted for his abstemiousness by saying he could not drink freely because of "the rebellion of his stomach."

Dr. Arnott, who saw Napoleon for the first time with Dr. Antommarchi in April, 1821, was informed that during the whole year of 1820 he had had nausea and vomiting occasionally with loss of appetite, and that his face had become very pale. Even so far back as the year 1817 the Emperor said he had been affected with pain in the stomach, nausea and vomiting, especially after taking food.

It was in September, 1818, that the British Government requested Napoleon's uncle, Cardinal Fesch, of Rome, to select a physician to proceed to St. Helena to attend Napoleon. Dr. Antommarchi, a Corsican, then prosector of anatomy at Florence, was eventually chosen. He was to be accompanied by a priest, and, according to Antommarchi, the man finally selected had just returned from twenty-six years' missionary work in Mexico. "He was full of zeal," he says, "but feeble, gouty and infirm; he could not possibly fulfil the object of his mission. A recent attack of apoplexy had also affected his tongue, and almost entirely deprived him of speech." So slowly did people travel in those days that Antommarchi did not land at St. Helena till September 18th, and did not see Napoleon till September 22nd, 1819, just a year after the question had first been settled. Napoleon became attached to this doctor, and left him, by a codicil to his will, a pension of £240.

As his disease progressed Napoleon had, besides the attacks of vomiting, which increased till the end, crises of agonising pain in the stomach and adjoining organs. During these attacks he would occasionally lose consciousness for some moments, and they left him in a state of extreme exhaustion. His fever was relieved, according to the treatment of the day, by copious bleeding, and the pain by hot baths, which soothed him.

He would not take any medicine and objected to a blister on the region of the stomach, but consented to a warm stimulating plaster being applied there.

Dr. Arnott, in his account of this last illness, says: "On the morning of April 27th (1821) our patient was low and comatose, had passed a restless night, and had had frequent attacks of vomiting, and on examining the basin, I observed that what had come off the stomach was a dark-coloured fluid resembling coffee-grounds, and very offensive. That vomiting continued until 3.30 p.m.; it then ceased and he went to sleep. Pulse kept at 84 and heat about natural. In the evening he was

more tranquil, free from pain, but low and exhausted."

The next day there was again perpetual vomiting, "a dark-coloured, grumous fluid, which contained small specks of blood."

From January, 1819, till May 5th, 1821, Napoleon was in constant suffering, not always severe, though at other times he would complain of pain like the cut of a knife. Towards the end of 1820 the disease became more acute, with attacks of intense pain, vomiting, chilled extremities, and somnolence. At the end of March, 1821, he was quite bedridden, vomiting blood. On April 18th he was conscious that the end was near, and exclaimed bitterly: "You have assassinated me slowly, deliberately with premeditation!"—the cry of a very sick and heart-broken man in exile.

On May 5th there raged over the usually peaceful little island of St. Helena a terrific and destructive thunderstorm, and when the sun set that evening the great ex-Emperor passed away.

Napoleon had left directions that a post-mortem examination should be made by his Corsican doctor, but stipulated that no English doctor should touch his body. The Governor humoured the dead man's whim, but ordered five English army and navy surgeons to be present and make a report, whilst the Frenchman conducted the examination. The body was fat, in spite of the weeks of vomiting, but Antommarchi stated that he was much thinner than he had been. The stomach was found to be the main seat of the disease, and in the roof of that organ was discovered a large, cancerous growth, extending over nearly the whole of the internal surface. There was also extensive local peritonitis, probably caused by the original ulcer.

The post-mortem report, signed by the five British medical officers, is as follows:

"On exposing the stomach, it was found the seat of extensive disease; strong adhesions connected the whole superior surface, particularly about the pyloric extremity, to the concave surface of the left lobe of the liver; and on separating these, an ulcer, which penetrated the coats of the stomach, was discovered one inch from the pylorus, sufficient to allow the passage of the little finger. The internal surface of the stomach, to nearly its whole extent, was a mass of cancerous disease, or schirrous portions advancing to cancer; this was particularly noticed near the pylorus. The cardiac extremity for a small space near the termination of the œsophagus was the only part appearing in a healthy state. The stomach was found nearly filled with a large quantity of fluid, resembling coffee-grounds."

In a letter from Dr. Arnott to Lieut.-General Sir Hudson Lowe, the Governor of the island, he further states: "The strong adhesions of the morbid parts of the stomach to the concave surface of the left lobe of the liver perhaps prolonged the patient's life; being over the ulcer, they consequently prevented the escape of the contents of the stomach into the cavity of the abdomen." There had obviously been some local peritonitis.

There was no evidence that the climate of St. Helena shortened Napoleon's life, nor that he suffered from epilepsy. His body showed scars of several wounds, especially on the thighs and legs, which he had carefully concealed at the time in order to give his troops the impression that he was protected by a supernatural star of destiny. He once told Dr. Warden that he had had eighteen horses shot under him in battles.

Another historical case still fresh in the minds of many of us is that of the Emperor Frederick of Germany, father of the present Kaiser, and husband of our own Princess Victoria. The account of his long illness, of his patient endurance of great suffering, is told by his medical attendant, Sir Morell Mackenzie, who was first summoned to see the then Crown Prince in May, 1887.

It appeared that the patient had suffered from what was supposed to be a catarrhal inflammation of the larynx in the previous January, and he himself attributed the beginning of his illness to a severe cold he caught in the autumn of 1886. The inflammation of the larynx had not yielded to ordinary treatment, and there was, in May, 1887, a small tumour in the left vocal cord, with no ulceration. A small piece of this tumour was snipped off and microscopically examined, but did not reveal the fact that it was malignant. At this time the Crown Prince's voice was little better than a gruff whisper, although it varied, being at times much clearer than at others, but by November of that year he had completely lost the use of it.

Although the microscopical examination of the growth had not proved it to be cancer, yet every one of the doctors in attendance on the case

suspected the nature of the disease, and the question arose as to whether the growth should be removed from the outside, but as this operation is dangerous to life and almost always destructive to the voice, and as the cancerous nature of the growth had not been proved, it was finally decided not to perform this radical operation.

Twice after this pieces of the growth were examined, but although it was not positively disproved, it was not certified to be cancer.

In June of that year some of us still remember the magnificent figure of the Crown Prince riding in his white uniform in the Jubilee procession of Queen Victoria, and there was no one in that splendid concourse who presented a nobler appearance or gave one more the impression of strength and power than this man, in the prime of life with the promise of so much in store for him. Yet death already had him in his grip and was daily tightening his hold upon him. In the early autumn there was a deceptive lull in the disease, but in October new swelling and ulceration began, which rapidly increased in size and intensity. The patient was moved to San Remo to avoid the bitter winter climate of Germany. An entire removal of the larynx was then suggested, and the question was submitted to the Crown Prince himself. His answer had of course to be written, as he had by this time completely lost the use of his voice. Sir Morell Mackenzie thus describes the scene:

"Professor von Schrötter discharged his unpleasant duty" (of acquainting the Prince of the verdict of the doctors) "with great tact and judgment in the presence of the Crown Princess and all the doctors. His Imperial Highness remained standing while the Professor was speaking, and received the terrible communication without any emotion. He was the calmest person in the room. We then withdrew, after placing the statement as to the operations on the larynx in the hands of the Crown Prince. In the course of a few minutes we received a communication from His Highness, written with a perfectly steady hand, stating that he declined to have his larynx excised, but would submit to tracheotomy should it become necessary."

In January, 1888, there was a decided change for the worse, and when Sir Morell Mackenzie was again summoned to San Remo, he found the patient in a precarious state, and the swelling interfered so much with the breathing that there seemed to be grave risk in postponing the operation of tracheotomy. This was therefore performed, and a tube was passed into the trachea. Some difficulty was experienced in getting a tube to fit which did not cause discomfort, and undoubtedly the patient had to suffer from too many different opinions and experiments, which probably only increased his suffering and hastened the end.

On March 9th, 1888, news came of the death of the old Emperor William, and the Crown Prince, now Frederick III, returned at once to his capital, although this was a serious risk to run considering the time of year.

Microscopical examination of the growth now left no doubt as to the cancerous nature of the illness. By April the Emperor had a daily rise in temperature, a large abscess had formed in the neck, and there was a steady decline in health. In May he rallied again, and was able to drive out. In June a false passage had formed between the larynx and the œsophagus, so that liquids taken by the mouth escaped through the tube in the throat.

Even at this time, in spite of increasing weakness and much suffering, the Emperor still insisted upon reading, signing documents, and receiving ministers and others, and the State work he accomplished remains a wonder to all who knew his condition. As the escape of food continued a feeding-tube had now to be used, through which all nourishment was given. On June 13th he developed inflammation of the lungs, which, mercifully, cut short his sufferings, and on June 15th "the eyes of the poor patient, which had languidly followed every movement of the Empress, became fixed, the intervals in drawing breath became greatly lengthened," and he was at last at rest.

Treatment.—When we come to consider the treatment of cancer I can only once more insist upon the fact that the early removal of the growth by the surgeon is the safest plan, and the more radical the operation the smaller the chance of a recurrence.

A very fair percentage of cases can be quoted where an apparent cure from cancer has followed early and free removal of the affected parts. But there must always be a certain number of cases where a cancer occurs in a situation which is inaccessible to the surgeon, and for these many different forms of treatment are tried, new methods are suggested, and are accompanied by a certain

amount of success, but many, unfortunately, have to be abandoned because they prove useless.

As to drugs, up to the present time no internal remedy has been found which will cause a malignant tumour to disappear, but certain drugs, such as arsenic, mercury and others, appear, in some cases, to retard the growth.

Caustics are now chiefly employed by irregular practitioners. The use of caustics, except in small and very superficial growths, cannot be recommended, and as the danger and pain from operations is now reduced to a minimum, there is every reason why the safer and more thorough method is preferred, for recurrence is less likely to occur after the knife than after caustics.

Sarcoma has been successfully treated by injections of a fluid, introduced by Dr. W. B. Coley, of New York, prepared from highly virulent cultures of the microbe of erysipelas. This is based on the fact that some tumours have disappeared after an attack of erysipelas.

With regard to the general management of patients suffering from cancer, the symptom which most requires treatment in advanced cases is pain, but it is satisfactory to know that pain is much diminished after the patients have been in hospital wards for a few days. Trained nursing, which means that they are made much more comfortable and that their wounds are soothingly dressed, accounts for the improvement. In old days, when morphia was given indiscriminately to sufferers from cancer, their pain was relieved for the time for some hours, but they became demoralised in their nervous system, so that they were unable to stand the slightest ache or even thought of pain, and so they craved more and more for the drug. In order to satisfy the patient the drug had to be repeated more frequently and in larger doses, until all appetite disappeared and emaciation and general debility were increased. To-day it is considered better to begin with mild sedatives, such as phenacetin and caffeine, or aspirin, and to leave morphia as a final relief.

There are, of course, exceptions to this general rule, for when the pain is excruciating morphia must be given early and two or three times a day.

Plain, easily digested food must be supplied, and a little alcohol to those who like it does no harm, and, on the contrary, helps them to enjoy their meals.

It is a relief to find in the general hopelessness and despair of cancer wards, that the spell is occasionally broken by the cheerfulness of some individual patient which penetrates the sad environment, and makes the nurses feel that the treatment, although it does not pretend to be a "cure," is not altogether in vain.

It must not be assumed that pain is a necessary accompaniment of early cancer; on the contrary, patients often lose valuable time by not consulting a medical man about a malignant lump which they think must be harmless because it is painless.

In 1895 Prof. Rontgen, the physicist of Wurtzburg, discovered what he called the X rays. They are a peculiar kind of radiation, produced in a vacuum tube on the passage of an electrical discharge of high tension from a Rumkhorff coil. The rays pass, with comparatively little absorption, through various optically opaque substances, such as wood, flesh, leather and clothes, which are therefore transparent to the rays. Bone and glass are semi-opaque, and some metals—lead and platinum—are almost entirely opaque to the X rays.

Since 1899 many patients suffering from rodent ulcer, a form of cancer which usually attacks the face, have been successfully treated by X rays. One specialist reports that of 125 patients to whom a satisfactory trial of the remedy has been given, 102 are free from the disease, while only two of them have exhibited a recurrence of the trouble, which in both instances was slight and yielded readily to further treatment. Of the twenty-three patients who were not cured, thirteen were the subjects of deeply seated, inoperable, practically hopeless cancer.

In the successful cases the average time for treatment and recovery was two months; the time occupied for each exposure varied from three to ten minutes, and the treatment was given daily or on alternate days, depending on the amount of local reaction caused.

The treatment of cancer by X rays is only local, for it does not affect in any way the growth of secondary deposits in parts of the body not under the exposure of the rays. Without injuring the skin it is not yet possible to administer exposure strong enough to arrest growth in the internal organs. Yet deep-seated pain can sometimes be relieved by it.

When the cancer is superficial treatment by

X rays almost always relieves pain, favours the healing of sores, retards the growth of the cancer, produces retrogressive changes, and even enables some patients to resume their ordinary life.

Even nodules under the skin may disappear without any affection of the skin.

We thus have a remedy which has perhaps done more to relieve inoperable cancer than anything hitherto known. But as yet we cannot tell beforehand how much any individual patient will be benefited, for the results after quite similar treatment may differ unaccountably.

It is believed that the rays must act on the cancer-cells and cause them to undergo some kind of degeneration, with subsequent absorption by the healthy cells surrounding the new growth.

X-ray treatment prolongs life in many patients suffering from external cancer, but it has very little, if any, influence on the growth of internal cancer, for a very thin layer of normal tissue suffices to protect cancer-cells from the action of the rays.

Trypsin and amylopsin are digestive ferments contained in the juice of the pancreas. The treatment rests on the theory that the growth of the cancer-cell depends on the presence of a ferment working in an acid medium, and that to check its development it is necessary to administer a stronger ferment in an alkaline medium. Yet occasionally cancer occurs in the pancreas itself, the organ which contains the very ferments which are supposed by some to be the natural enemies of cancer.

During 1906, 209 cases (64 males and 145 females) of malignant disease were examined microscopically, and eleven cases were subjected to treatment by trypsin, according to the instructions of the physicians who had recommended this method of dealing with cancer, but the committee reported "that the course of cancer, considered both as a disease and as a morbid process, is unaltered by the administration of trypsin and amylopsin."

Six of the eleven patients underwent a course of treatment varying from 66 days to 118 days, and it was found that the treatment did not prevent the patients from losing weight. The treatment entailed large injections under the skin, which were much disliked by the patients.

In 1903 hopes were aroused in Paris and elsewhere by the announcement of Dr. Doyen, a dexterous surgeon, that he had discovered the long-looked-for microbe of cancer, and that cultures could be made from it to inject into human patients, with the object of retarding the growth or even causing it to disappear. Alas! When the method was tried in this country on a series of cases, most of which had been seen by Dr. Doyen and pronounced by him to be suitable for the supposed cure, it proved to be a dire failure. "In none was there any improvement," and the treatment had to be suspended. In some of the cases the injection was followed by severe pain, and in others it caused alarming symptoms of collapse.

I think you will be interested if I tell you quite briefly of the foundation of the Cancer Charity of the Middlesex Hospital, an account of which has been compiled by Dr. Sydney Coupland, consulting physician to the hospital.

The first ward of twelve beds was opened in 1792 in accordance with resolutions passed by the Governors. One of these resolutions provided that the patients should remain an unlimited time, "until either relieved by art or released by death, unless it should be thought necessary to discharge them for ill behaviour."

From that year till now, a period of 117 years, the cancer department has remained a distinctive feature of the hospital, having its separate foundation and affording a refuge to a vast number of unfortunate persons. Benefactions received from time to time have enabled the single ward to be multiplied into four.

At a visit which I was allowed to make a month ago I found three female wards in a separate wing, besides a male ward—forty-nine beds in all. I learned that the average time occupied by any one patient is four months, and that the beds are all free. Cases which can be benefited by surgical interference are at once so treated, but most of the patients are inoperable, and are carefully tended by doctors and nurses until they go to another world. There are about six deaths every month, and there is always a waiting list of at least a score to fill the vacancies. Pleasant sitting-rooms are provided and musical and other entertainments take place at frequent intervals.

At the inauguration of the Cancer Wing it was pointed out that Lord Bacon had observed that medical men should make themselves proficient in

physic by studying one disease at a time, and it was suggested that by confining one or more wards to cancer only, the attention of doctors would be directed strictly to the study of this disease.

"They will see facts as they arise in the aggregate from a large number of patients; new lights will appear and new discoveries will probably be made."

The eminent Dr. Sydenham, the English Hippocrates of the seventeenth century, once wrote: "I have often thought that if I knew accurately the natural history of any disease, I should never be at a loss for a proper method of treating it."

The original funds were supplied by an endowment from Mr. Samuel Whitbread, after whom one of the wards is appropriately named, though his name was not disclosed until after his death.

One of the main objects in view was to institute trials of new methods of treatment, when properly authenticated, and when the nature of the remedy has been communicated to the physician and surgeon in charge of the patient.

Amongst the favourite "cancer cures" in all ages has been the use of caustics, which have been vaunted as sufficient to destroy the morbid growth without using the knife. Arsenic had been so employed by the Greeks and Romans and again in the middle ages. It was the chief ingredient of Guy's caustic and Plunket's paste, which killed Lord Bolingbroke and many others. Lord Arundel's cancer cure, the receipt of which he bought from the ignorant wife of a blacksmith, consisted of arsenic and corrosive sublimate, which sometimes removed an external cancer but usually destroyed the patient.

In 1880 a trial was made of Chian turpentine, which a surgeon had maintained had a remarkable effect in checking the growth of local cancer, but it was found that no result at all was produced by the administration of the drug.

The record of trials is one long list of failures.

When Lord Metcalfe, the Governor-General of Canada, returned to England in 1845, dying of cancer, he was pestered by offers of cures. Italian professors, mysterious American women, learned Germans and obscure Irish quacks, each pressed forward with an infallible remedy. Besides baths, mesmerism, and homœopathy, he was promised restoration to health from an application of the common dock-leaf; an infusion of couch-grass; a plaster made of rose-leaves, olive oil and turnip juice; a plaster and powder in which some part of a young frog was the principal ingredient; a mixture of copperas and vinegar; an application of pure ox-gall; a mixture of Florence oil and red precipitate, etc. Another local application much in favour was a poultice of carrots, but it was found that one of red onions, mashed and applied cold, was too irritating for use.

In 1900 laboratories were opened at Middlesex Hospital in connection with the new Cancer Wing for the systematic scientific investigation of cancer. Here every day are working Dr. Lazarus Barlow and ten assistants in different rooms, all busily employed in trying to solve the various mysteries connected with the disease.

When the cloud is lifted from obscurity we may be fairly certain that the microscope will be our most valuable ally. Other countries have their workers, too; Denmark, France, Germany, and the United States, all have laboratories devoted to cancer investigation, and similar work is just being inaugurated in Belgium, Spain and Italy.

In the second Report of the Middlesex Laboratories an account is given of some empirical methods of treating inoperable cancer between 1900 and 1902. No improvement occurred after the administration of thyroid extract. Negative results were also obtained from the application of electric currents of high potential and rapid frequency, though it was found that cancer patients, like others, improved in tone and general nutrition. "The patients feel better, their appetite is increased, and for a while, at any rate, all seems to go well with them."

Even less satisfactory were the results obtained by treatment with cancroin, which was highly praised in Vienna as a method of destroying cancer-cells by a process of fatty degeneration, after which the disease was supposed to disappear by absorption or expulsion. The general public are said to believe that the cure for cancer, when it is discovered, will prove to be something very simple, so patients have been treated by applying to their wounds lint soaked in hot water and violet-leaves, an ointment made from the petals of the flowers of an African marigold, strong ammonia, molasses made from the sugar cane, and aniline chloride, all of which had the reputation of having cured real or supposed cases of cancer.

In 1903 nine cases were treated, with their own consent, at Middlesex Hospital, by a treatment recommended by Dr. Otto Schmidt, of Cologne, who claimed that he had discovered the parasitic cause of cancer, and that from this parasite he had been able to manufacture a remedy. Dr. Schmidt himself visited the hospital and expressed himself satisfied with the cases chosen for the test, but unfortunately it was found that the treatment in no way modified the course of the disease and none of the patients derived any benefit.

The fourth report gives statistics of the ages of 6732 cancerous patients admitted to the Middlesex Hospital from its earliest records in 1746 until the end of 1904.

In analysing the cases which occurred in the alimentary tract, it was found that the numbers in males exceeded by about seven times those in females, and also that the age when the discovery of cancer was made was later in men than in women.

Cancer of the breast constitutes fully one third of all cases of female cancer; it may occur as early as 22 years or as late as 89 years, the most usual age being between 40 and 60. Amongst men cancer of the breast occurred in twenty cases, varying in age from 40 to 86. Roughly speaking, it may be said that cancer in both sexes occurs early in the organs peculiar to the sex, while later cancer is manifested in the skin, and the intermediate group is shown in the alimentary system.

There is a general tendency for men to be affected by cancer at a later age than women, the male average being 55·2 years compared with the female 49·9 years. Cancer under the age of 35 years is commoner in women than in men. The youngest case recorded was that of a male under 20 years of age, while of the whole number of 6732 cases there were twenty-one others between the ages of 20 and 25.

Radium, the element discovered by the electrical experiments of M. and Mme. Curie in Paris, is obtained from pitch-blende residues in Bavaria, Cornwall, and other places. A freshly prepared radium salt has its energy stored up and reaches its highest power in about three weeks, and then seems to keep it indefinitely. Its market price is £10 per milligramme at the cheapest, or £300,000 per ounce, always supposing that you could find an ounce in the world to buy.

Paris already has a Radium Institute, and one on similar lines is now to be erected in London, with H.M. the King as Patron. The institute, by the generosity of Lord Iveagh and Sir Ernest Cassel, has acquired radium to the amount of five grammes —about a teaspoonful. The treatment will be strictly limited to that by radium or other radio-active substances; the poor will be treated free, but the well-to-do patients must pay fees.

The chemical properties of radium are remarkable: it decomposes water into hydrogen and oxygen, and converts oxygen into ozone, it turns glass a violet colour, makes salt blue, and rots cloth. The rays emitted by the highly active preparation discharge a charged gold-leaf electroscope even at a distance; they destroy the vitality of seeds.

Radium gives off heat, its temperature being always slightly higher than the surrounding air; it will melt its own weight of ice in an hour.

The discovery of radium is perhaps the most revolutionary of modern science, but there is a great deal yet to be learned about it, chemically, physically, and medicinally. As yet we are only in the experimental stage with an expensive substance, only one part of which can be obtained from 10,000,000 parts of the mineral containing it. When we are told that radium is formed from uranium we are reminded of the labours of the old alchemists, who spent their lives in trying to convert baser metals into gold and silver. Already we are warned that the marvels of radium will be utilised to advertise quack remedies in order to deceive the public. They will claim to emit radium rays. This is a serious danger, and care must be taken to prevent the use of the name "radium" for anything which does not contain the substance itself.

There is no doubt that radium, like the Röntgen rays, has a powerful influence on morbid cells, though the influence is also exerted on cells which are perfectly healthy. The essential points which now require investigation are—the application of radium to deep internal cancers, as by introduction of a tube into the middle of the tumour, and the conditions which will insure the healthy structures remaining undamaged while the malignant tissues are being treated.

Sir Frederick Treves states that radium can effect a cure when X rays cannot, and mentions cases of old-standing and deep-seated rodent ulcer which have been cured at the Paris Institute "by two sittings of radium lasting one hour each, the parts being finally left free from attachment to the deeper parts, the skin being soft and pliable."

Similar astonishing results have been attained in this country by the few who have been in the possession of radium since 1903.

If you put a penny into a closed glass vessel in which there is already a tube containing radium, the penny becomes radio-active and remains so when separated from the radium. The penny loses the radio-activity when it is rubbed with emery powder or washed in nitric acid. But the radio-activity is not lost—it has been transferred to the emery powder or the nitric acid. Radium thus has the power of shedding a sort of vapour which renders everything it touches radio-active. This radio-activity is of at least three different kinds, one or two of which can be separated from the group, but no one yet knows which of the different kinds is of value in treating cancer.

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Iliac Constriction in Intestinal Stasis.—Mr. ARBUTHNOT LANE demonstrated at Guy's Hospital on a case of chronic intestinal stasis in which the symptoms were supposed to be due to disease of the appendix that they resulted from a reduction of the calibre of the lumen of the ileum within two inches of its termination in the cæcum. This condition of constriction of the ileum has been described by Mr. Lane on many former occasions, but it seems to have escaped the observation of surgeons, in spite of the facility with which it can be demonstrated and dealt with. This constriction is brought about in the following manner. The loaded cæcum tends to sag downwards and inwards into the floor of the pelvis. To oppose this tendency peritoneal bands are formed, which are attached to the cæcum and extend upwards and outwards to the abdominal wall. These bands are usually attached to the appendix, commencing to grip it from its entry into the cæcum, and gradually extend along its length. In this manner a portion of the appendix is converted into a ligament or tie opposing the downward displacement of the cæcum. Unfortunately, the appendix, being hollow and secreting, the kink which develops at the junction of the proximal anchored part with the distal mobile portion results in the distension of the distal segment and the development of so-called appendicitis in its varying forms. The peritoneal bands so anchoring the appendix may be quite distinct from its normal mesentery, or they may be blended with it. Their division leaves the appendix normally mobile. In many instances, however, the appendix may not become involved in adhesions, and may be quite free. While much of the added weight of the cæcum is borne by the adhesions already described, the strain which is transmitted towards the middle line results in the development of bands which form on the under surface of the mesentery chiefly about two inches from the cæcum, fixing the ileum at this point and making its terminal two inches perform the purpose of a ligament supporting the cæcum in addition to its normal function of drainage. This fixation of the ileum produces a constriction of its lumen and a consequent obstruction to the passage of fæces through it. Clinically the symptoms are tenderness below and a little to the right of the umbilicus and quite apart from the appendix, unless the latter is involved in adhesions, when its tenderness can be clearly defined as distinct from the other. The stasis of the contents of the small intestine produces flatulent dyspepsia with pain. This constricted condition of the small intestine is the cause of much disappointment in cases of operation for appendicitis, since after the removal of a mobile appendix or of a fixed and inflamed one, part or the whole of the pain and discomfort remains, much to the disappointment of the patient and the vexation of the surgeon.

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CONTENTS.

PAGE

***A Clinical Lecture on a Case of Endosteal Sarcoma.** By H. J. WARING, M.S., F.R.C.S. 17

***Interesting Cases of Abdominal Surgery, including some Rare Forms of Intestinal Obstruction.** By HENRY CURTIS, B.S., F.R.C.S. 22

***On some Affections of the Eye-Muscles.** By A. HUGH THOMPSON, M.A., M.D., B.C. 29

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A CLINICAL LECTURE

ON

A CASE OF ENDOSTEAL SARCOMA OF THE TIBIA.

Delivered at St. Bartholomew's Hospital.

By H. J. WARING, M.S., F.R.C.S.,

Surgeon to the Hospital, and Consulting Surgeon, Metropolitan Hospital.

GENTLEMEN,—The patient I shall discuss is a man who came into the hospital a short time ago on account of what may be roughly called an affection of the knee. I am using this term in popular language, because there are one or two matters of importance in connection therewith which will soon become apparent. The case in reality is an example of that somewhat uncommon affection called "endosteal sarcoma," and this is the disease about which I propose to lecture to-day. The patient, whom you see before you, is a man, æt. 36 years, a joiner by occupation. He came to the hospital on October 30th, 1909, complaining of a swelling in the region of the left knee, and gave the following history, many points in which are of considerable clinical importance. In October of 1905 he experienced pain in the region of the left knee-joint, and for this sought medical advice and was examined. He was thought to have an "*affection of the knee-joint*," and for this was given an elastic support. This support he wore for six months, and during the whole of this period he followed his ordinary occupation, that of a joiner. After wearing this support for six months he found that he was not materially benefited by it, and discontinued its use in March, 1906. From this date until February, 1908, he appears to have gone on with his work, although he suffered some difficulties as regards the left knee, the difficulties being mainly in the nature of pain, but not sufficient

to prevent him following his occupation. In February, 1908, a swelling was noticed in the region of the left knee-joint, but rather below it, when, acting upon the advice of friends, he visited a well-known London "*bone-setter*," with the intention of having his knee attended to. The "bone-setter" investigated the condition of the knee, and I am told by the patient that this "bone-setter" diagnosed the affection as "a displaced cartilage of the left knee-joint." This is the first occasion apparently that there is any evidence of anything like a definite diagnosis having been made. Immediately afterwards, that is to say in February, 1908, he was anæsthetised, and the knee-joint forcibly bent and moved, with the intention of breaking down adhesions within this supposed affected knee-joint. The next point of interest is that three weeks later the patient was again anæsthetised and similar treatment carried out, that is to say forcible bending and manipulation of the supposed diseased knee, or the knee affected with a displaced cartilage. For subsequent manipulations and treatment this patient visited the "bone-setter" on fourteen different occasions, but only on two occasions was the administration of an anæsthetic necessary. Thus the patient continued regularly attending and being treated by the "bone-setter" from February, 1908, until the latter part of October, 1909. The region of the left knee-joint, the patient says, apparently continued to increase in size. The patient was now seen by another medical man, who aspirated the swelling and drew out from it what was stated to be a quantity of blood-stained fluid. The diagnosis was then made that the patient had probably got some form of growth in connection with the left fibula, and on this account he was sent to me at the hospital for investigation and treatment. What I have given you up to the present time, gentlemen, is mainly what may be said to be the patient's history, that is to say, the clinical events and facts of importance which have taken place from the commencement of the patient's disease up to the end of October, 1909. For a moment I will refer to the patient's past. There is nothing of importance in that; I will merely say that he has not suffered from injuries, and apparently has had no diseases. Concerning the family history, the patient is married and has two healthy children. His father is living, æt. 62 years, and his mother died æt. 47 years. The man has two brothers and five sisters still living, all of whom are in good health. One brother died, æt. 25 years. The only point of importance is that there is no history of the occurrence of any malignant disease.

Now we come to the period when the patient came under my observation. He was able to walk perfectly well when he came to the hospital, and had been following his occupation, but up to this time he suffered from occasional pain in the region of the left knee-joint. When I first saw him I noticed that he had a well-marked swelling situated in the region I show you, overlying the upper portion of the shaft of the fibula and a little below its head. As one looked at the region of the knee-joint one could see that the affected limb was very much wider from side to side than the sound one, and there was a well-marked boss in this position. On taking measurements of the affected leg it was found that the circumference on the diseased side was $15\frac{1}{2}$ in. at the level of the most prominent part of the swelling, and on measuring the sound limb at a similar level it was found to be $12\frac{1}{2}$ in. Thus there was a difference of 3 in. Concerning the movements in the knee-joints, they were perfectly free, both active and passive, and during ordinary movements the patient apparently did not suffer any marked pain. Latterly, when following his ordinary occupation, he has experienced considerable pain in the portion of the leg immediately below the knee, but not in the joint. On palpation of the swelling in the region of the upper part of the tibia, it was noted especially on the superior and internal aspect that on firm digital pressure a distinct sensation could be felt, a sensation which is given two names—parchment crackling or egg-shell crackling. Probably you can imagine what it is like, since it resembles the effect of exerting pressure upon an egg, so that it becomes cracked in numerous places. When he first came under observation this "egg-shell crackling" was very marked, but soon it could not be readily detected, on account of the frequent examinations which were made. On investigation of the other parts of the patient we found there was no evidence of enlarged lymphatic glands, or of visceral swellings in any part of the body, and at that stage of the examination we made the diagnosis that the patient was suffering from "endosteal sarcoma" arising from the "interior" of the upper extremity

of the left tibia, breaking through on the outer side and overlapping the fibula. The reason we thought it was much more likely to be in the tibia was on account of the increased width of this bone, although the most apparent prominence was in connection with the fibula. X-ray photographs were next taken of the affected portion of the limb both in an antero-posterior direction and from side to side. These I will now show you. The first is a skiagram taken in the antero-posterior direction, and in it you can see the outlines of the femur, tibia, and fibula, and if you look at the femur in detail you find the outline is practically unaltered. The same remark holds with regard to the fibula itself. But apparently there is a shadow which suggests something lying superficial to it. When we examine the tibia, however, in the upper portion, we find it is much wider from side to side than it should be, and there is a well-marked shadow occupying the upper fourth of the bone, extending as far as the articular cartilage of the knee-joint. There are certain processes running inwards in connection with that shadow which are suggestive of bone. The external layer of compact bone has become thinned, and this is the explanation of the egg-shell crackling.

When an endosteal sarcoma develops in the interior of a bone it gradually increases in size, occupies the medullary cavity of the affected portion of bone, and causes thinning of the dense or compact bone on the surface, until the latter becomes so thin that one can make an impression upon it with the finger. It is this which gives rise to typical crackling. It is a thing which you will only learn to recognise by practical experience; it is difficult to describe it. So far with regard to investigation by X-rays in an antero-posterior direction. The skiagram taken in the other direction merely shows that the fibula is not involved. Thus the examination by X-rays has practically confirmed our diagnosis, namely, that the patient was suffering from some form of endosteal growth, occupying the interior of the upper portion of the tibia and breaking out on the external aspect so as to overlap the fibula. We have now been able to make an absolute diagnosis, or at least as far as one can, without actually taking out a portion of the swelling and seeing it by naked eye and under the microscope.

Endosteal sarcomata occurring in the ends of long bones are, in the majority of cases, what are known as myelomata or myeloid sarcomata, that is to say, they are growths which histologically contain a large number of multinucleated cells mixed up with a large number of spindle-shaped or round cells. The one point which in this case is favourable of myeloma is the duration of the patient's disease. When I began to speak I laid stress on the fact that this patient's disease first became apparent in February, 1905, and the disease gradually went on giving rise to increasing symptoms until October, 1909, that is to say, four and a half years. From the point of view of sarcomata in general that is a very long history. As a general rule, in ordinary sarcomata in connection with bones, if they are not treated by surgical measures, the duration of life of the patient is very much less than that. I think one may say that after sarcoma of the long bones has definitely made its appearance, in the majority of instances eighteen months to two years sees the end of the patient unless surgical treatment has been carried out. But in this case it has taken four and a half years to arrive at the stage at which you see it, and that is strong reason for saying that this patient probably has myeloma, or myeloid sarcoma. There is a good deal of difference in connection with these two terms in the text-books, and I mention this because some pathologists assert that myeloma is a non-malignant disease whilst others maintain that it may become malignant. It is difficult to be very dogmatic on the point, because it undoubtedly happens that a certain number of tumours in connection with long bones which, to begin with, have definite symptoms of a myeloid growth, later take on typical sarcomatous characteristics, and I have seen this happpen several times. Therefore I feel very doubtful about saying that myeloma is always a non-malignant disease: I do not think it is.

Having made this diagnosis, and taken into consideration the points I have mentioned, that the entire thickness of a segment of the tibia was involved, and that the growth had burst through the bone on the outer side and had overgrown the upper portion of the fibula, I advised the patient that the best course to pursue in his case was to have his limb removed above the level of the disease. The growth was situated in the *lower limb*, and there were practically four inches of bone

involved in the disease. It is, however, occasionally possible in the case of so-called myeloid sarcoma, where the disease is localised, to perform resection operations and leave a useful limb. This is especially the case in connection with the bones of the upper extremity. Here is a skiagram of a patient who had one of these myeloid sarcomata in connection with the lower part of the radius, which was successfully treated by local removal. The affection was confined to a bone in the "*upper limb*," and in this case resection operations are much more useful, since if the patient can move the fingers afterwards he has a much more useful limb than when fitted with an artificial hand. In the lower limb it is more difficult to perform these resection operations and leave a useful limb afterwards, especially in the artisan class. If, in this particular patient, the disease had been localised to the interior of the bone, and not extended to the adjacent tissue, it might have been possible to resect the upper portion of the tibia, to take away the articular surface of the femur, to slide up the entire limb and fix the fibula to the outer aspect of the femur, and also to fix the lower portion of the femur to the upper part of the excised tibia. This would leave a limb four to six inches short, with a stiff joint, and not a very strong one. It is doubtful whether it is more satisfactory to have such a limb or to have a well-made strong artificial limb, which in all probability would be more useful. But one important reason against attempting a resection operation in this patient was that the growth had broken out on the outer side, and was evidently extending into the surrounding tissues.

For these reasons I recommended that this patient should have an amputation performed so as to give him a useful limb, one which would be best for him, remembering his occupation, that of a joiner, and at the same time give him the least likelihood of recurrence. This operation was done, and the patient quickly recovered. I have had him brought in to-day so that you might see what type of man he is and the result. Here are histological sections of the growth, which was removed, and also the limb. The limb has been sawn across, and you see the tibia occupied by growth, in one place broken down and containing a blood-cyst. You also see the growth extending outwards over the fibula. It is a very interesting specimen from many points of view, and I will send round for you to compare with it a section through the limb of a patient who had a periosteal sarcoma. In one the bone is distended and occupied by the disease, whilst in the other the bone is normal as regards its main outlines, the growth being on the surface and extending into the surrounding tissues. The endosteal form has commenced in the interior of the bone and expanded it; the other, periosteal, on the surface and involving the bone, growing round it and extending into the surrounding tissues.

Now as regards the particular clinical points or facts of diagnostic importance which we may draw from this patient's case. The first deduction we may draw from it is this: that when a patient suffers from pains in the region of the knee-joint and which he refers to the knee-joint, do not straight away assume that the knee-joint is the seat of disease. The symptoms may be caused by something wrong in the neighbourhood of that joint. In this case it was something immediately below the joint, an endosteal sarcoma of the tibia. A similar mistake is not uncommonly made in connection with patients suffering from acute osteomyelitis of the lower part of the femur or the upper part of the tibia. The great majority of these cases, on account of the pain, say that they have rheumatic pains in the knee, and not infrequently, if the medical man is a little careless as regards inspection and investigation of the parts involved, he is liable to treat such patients for rheumatism by medical measures, and to be a little startled later on that there is something more there than he expected. This is another disease entirely, but the remark holds good as regards this particular condition. Whenever patients come complaining of suffering from painful affections in the region of the knee, do not be satisfied by merely looking at the knee-joint itself, but investigate the parts above and below, and then you will avoid making a mistake such as was made originally in the case of this patient. The second important point as regards this patient was this: that if the disease had been diagnosed at a sufficiently early stage, before the entire thickness of the bone had become involved, and before it had burst through and involved the surrounding tissues, it is very probable that a local operation would have been of value, that is to say, one could have removed the affected segment of the tibia by a local excision operation,

and the patient would have survived with a practically sound limb. These are the two main points which I wished to impress upon you in connection with this patient—that in patients who are suffering from affections especially in connection with, or in the neighbourhood of, the knee-joint, be careful to investigate the parts above and below in addition to the joint itself, and do not trust your patient's statements with regard to the position of the trouble; because a patient says he has disease of the knee do not think that he has disease of that joint; it may be something else which you may pass by if you are not on the alert. In cases of this kind examination by X rays will enable you to see the extent of disease, and the condition of the adjacent bones and joints; therefore in cases of doubt, and when a diagnosis is not clear, I strongly advise you to have an X-ray photograph taken of the affected portion of the limb before making a diagnosis for treatment. I have no hesitation in saying that if this patient had been examined in this manner in 1905, the true nature of the disease would then have been ascertained, and in all probability a local excision operation would have been sufficient, and the limb have been saved.

I now wish to refer to a few general points concerning endosteal sarcomata. At present I have simply used this patient as an illustration of a type of disease. Endosteal sarcomata, as I have already mentioned, are of two varieties. First, there is what is called "*myeloma*" or "*myeloid sarcoma*" in which the tumour which forms in the bone consists in the main of multinucleated cells with a number of round and spindle-shaped cells between. Under the microscope you will see a typical example of that disease. It is the ordinary variety of myeloma. Secondly, do not forget, in addition to that disease, all the ordinary varieties of sarcomata may commence in the interior of a bone. Further, do not assume because a sarcoma primarily begins in the interior of a long bone that it is necessarily myeloid; it may be the round-celled or spindle-celled variety of sarcoma. In addition it is very important to remember that when myeloid sarcoma has existed for a long time it may take on the characters of a round or spindle-celled sarcoma, and then it grows quickly and infiltrates the surrounding tissues, and causes secondary growths in the lymphatic glands and the internal viscera. I have seen this happen on several occasions. When tumours of this kind have been investigated histologically they have been found as regards portions to have the microscopical characteristics of a typical myeloma, and in others those of typical round- or spindle-celled sarcomata.

Concerning the bones which are particularly liable to be the seat of myeloma or myeloid sarcoma, I think the tibia is the commonest, and the upper extremity of the tibia. The situation of the disease in this patient happened to be one of the most common for the disease. This particular disease is not common. When it does occur it affects the cancellous extremities of long bones, and the long bones which are especially liable to be affected are the tibia, radius, ulna, the lower extremity of the femur, the fibula and the humerus. Occasionally the clavicle is involved. I have seen one or two cases in which this disease occurred in the clavicle. As regards the bones involved, one would think as this disease occurs in the cancellous tissue it would be especially prone to affect the short bones, such as the carpus, tarsus, and bodies of the vertebræ. But as far as my memory serves me I have never seen a case in which a patient had myeloma or myeloid sarcoma of the bodies of the vertebræ. I have seen this disease in one case commencing in the os calcis, but that is the only short bone which I can call to mind seeing the disease in. As regards prognosis in connection with these growths, in this particular patient where the limb has been removed, and where there is no very definite evidence that there is spindle-celled growth and infiltration of the adjacent tissues, I think the prognosis is very good, both as to local recurrence in the stump and secondary growths elsewhere. Pure myelomata, I think, never give rise to secondary growths, at least, I have never met with a case in which they occurred. When the disease is met with in the early stage and it is removed by local excision, the prognosis is quite good.

These are the main points to which I wished to draw your attention in connection with this patient. There are a few specimens of similar growths under the microscope, as well as the skiagrams for your examination.

April 18th, 1910.

INTERESTING CASES OF ABDOMINAL SURGERY, INCLUDING SOME RARE FORMS OF INTESTINAL OBSTRUCTION.*

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GENTLEMEN,—Shortly before your hon. secretary, Dr. Cardno Still, conveyed your committee's suggestion to read a paper, I happened to have seen in consultation with a member of that committee, Dr. Gibbes-Smith, Medical Officer of Health at Teddington, two cases of intestinal obstruction, which have happily survived the necessary "surgical interference," as it is sometimes irreverently styled; and in responding to the invitation with which you have honoured me, these two interesting cases naturally suggested the subject for our consideration to-night.

I shall, however, only briefly allude to these cases of Dr. Gibbes-Smith.

One was an old lady, æt. 79 years, with an old, but reducible left inguinal hernia, and a large mass of what appeared to be glands in the right groin; so that the diagnosis of the cause of the obstruction present was not obvious until on opening the peritoneal cavity a right femoral hernia was found, a small portion of the lumen of loop of bowel being tightly nipped at the internal orifice of the crural canal, and held there firmly by a band of tissue leading down and out through the canal to the deeply congested enlarged glands in the groin. Division of the band released this Richter's femoral hernia, the enlarged glands were removed, and a cure resulted. This illustrates the fact that, as Rose and Carless put it, "the intestine above the site of the strangulation becomes paralysed, and peristalsis is entirely arrested even in a Richter's hernia," where only a portion of the lumen of the bowel is involved, so that actual interference with the passage of fæces is not an essential for the condition known as "strangulated hernia."

The second patient was a young married woman, æt. 37 years, in whom complete obstruction was found due to a malignant growth in the anterior wall of the sigmoid colon, very low down, so that it could not be felt from above or below; as it was found to be in close contact with a large growth in the left ovary, it may have been an instance of extension by direct continuity. The prognosis being so grave it remained only to perform left inguinal colostomy.*

Another interesting case was seen shortly afterwards at New Cross, in consultation with Dr. Albert Pryse. The patient was a man, æt. 79 years, suffering from great distension, retching, and complete obstruction.

Digital examination of the rectum enabled one just to reach a tight stricture, fixed to the sacrum, but rather more pronounced anteriorly than posteriorly.

Though colostomy was performed the patient died the following day, when an extensive growth was discovered originating in the mucous membrane of the trigone of the bladder, extending, secondarily, to the top of the prostate and the rectum. The appearances negatived its having originated in the prostate. The pelvic peritoneum was studded with small growths. The only history of urinary trouble noted by the patient, strange to say, was that a catheter had on one occasion, twelve months previously, been passed for retention; since then there had never been any symptom to suggest bladder trouble.

Since my return from South Africa I have recorded certain cases of abdominal surgery occurring amongst both whites and natives, and this evening I propose to recount a further series of cases which, occurring entirely amongst Matabele natives admitted under my care, will, I hope, seem sufficiently unusual to warrant wider publicity than a brief record in the 'Reports of the Bulawayo Memorial Hospital,' which I prepared for the years 1904–6.

The series comprises the following cases:

(1) Penetrating wound of a hernial protrusion

* Read before the Richmond Division of the British Medical Association.

* At the discussion on this paper, the writer was informed that the patient had died about seven months later, two or three days after an exploratory operation had been performed by a well-known London gynæcologist, in the hope that the tumour of the ovary was, after all, benign, since after the original colostomy for some time the general health of the patient had greatly improved—so much so that her weight had temporarily increased by two stones. The second operation confirmed the utterly hopeless condition of things.

(self-inflicted), the intestine and mesenteric vessels being transfixed.

(2) A large umbilical protrusion, simulating a reducible hernia, found to be a trabeculated sac, containing only ascitic fluid.

(3) Tubercle of the extra-peritoneal tissue of the anterior abdominal wall, associated with pleurisy and adhesive peritonitis (polyorrhomenitis).

(4) Volvulus of the small intestine, secondary to a hernia into the duodeno-jejunal fossa.

(5) Volvulus of the cæcum, from chronic peritonitis, and strangulation of the descending colon by a traction diverticulum.

The consideration of the present series of admittedly rare or unusual cases will not be without advantage if it stimulates us always to keep in mind possibilities, as well as probabilities, in any given case, simple or complex, so that the significance of the symptoms and signs, and their grouping, however familiar, or the contrary, may be grasped as early as possible.

"Even Homer sometimes nods," and the most alert of us sometimes become blind to the significance of symptoms and signs with which we were perfectly familiar at an earlier period of our professional career. Mistakes may thus arise, not so much from a lack of knowledge as from the fact that the possibility of such and such a condition has "never once crossed the mind."

Such an obvious truism is submitted with the utmost diffidence and respect, as much as a reminder to oneself as to his audience, since, whether engaged in consulting or general practice, we all, even in this democratic age, live in palaces of glass, where it is unwise to throw stones!

Preliminary observations.—Probably more often than not an exact diagnosis in the case of intestinal obstruction is only arrived at after an exploratory laparotomy. Nevertheless a few general remarks dealing with some of the commoner symptoms of obstruction, which will remind you of well-known facts, may serve as a useful preliminary to the perusal of the record of this series of cases.

From the practitioner's point of view the essential point to bear in mind in dealing with a case exhibiting possible obstruction symptoms is to avoid merely symptomatic treatment. For instance, a patient may come to you convinced that he is only suffering from constipation, with or without hæmorrhoids. Further investigation, which should always be performed in a definite and systematic way, may afford conclusive evidence of intestinal obstruction, high or low.

Again, if summer diarrhœa happens to be prevalent and a child is brought with symptoms of muco-enteritis, do not hastily conclude that it is suffering merely from a more or less temporary gastric disturbance. Examination of the abdomen and bowel may reveal the presence of an intussusception,* and the earlier this is discovered the greater the chance of successful treatment.

On the other hand, vomiting, often merely the result of indiscretions in diet, liquid as well as solid, may signify by its special characters—gushing, and having no relation to food—not abdominal disease, but intra-cranial mischief, urgently needing relief of the tension, *e.g.* cerebral abscess. When vomiting results from intestinal obstruction it is an equally urgent signal.

The cardinal symptoms of intestinal obstruction.—Intestinal obstruction may be acute, or chronic, and chronic obstruction may suddenly become acute.

The cardinal symptoms of obstruction are a history of constipation of long or short duration, abdominal distension, with a variable degree of pain and vomiting, which, however, may appear only late, or be hardly noticeable.

Vomiting, as you will remember, comes on early in acute obstruction high up, and is provoked if food is taken in this condition. In making a diagnosis do not be deluded into waiting until the vomit has become fæcal and offensive, for this condition only indicates obstruction low down or a late stage of obstruction high up. In either case delay is dangerous. Where the obstruction has been gradual vomiting may not appear for weeks, as Berry † and others have recorded.

In cases of acute volvulus of the small intestine vomiting is commonly present, and its absence in Case 4 of the present series is therefore noteworthy.

In all cases of strangulation, internal or external, delay in relief involves the grave risk of death from reflex shock and septicæmia, the result of compression of the loop of bowel and the corresponding nerves and vessels, which may lead to actual gangrene and septic peritonitis; and in obstruc-

* See Berry, "Surgical Diagnosis."

† *Ibid.*

tion from stricture there is the same danger of gangrene and perforative peritonitis.

Where there is a stricture, the bowel proximal to it not only becomes distended but hypertrophied, and the efforts to force its contents past the obstruction become manifest through the abdominal wall as a visible peristalsis in the distended portion. This is a most significant hint, but so long as it is present we may assume the absence of acute peritonitis.

The onset of peritonitis in such a case is indicated not only by the patient's general condition and facies, and the increasing pain and other symptoms, but by local or general rigidity of the recti and other abdominal muscles, and cessation of visible peristalsis. These are very grave signs; the patient's condition is critical, and an operation to relieve the obstruction and drain the peritoneal cavity of any effusion affords the only chance of recovery.

Perforative peritonitis under such circumstances commonly has its seat of origin in the cæcum, and of this I have recently had two instances where gangrene of the cæcum necessitated right inguinal colostomy, and drainage of the peritoneal cavity.

One patient, a very feeble old man, æt. 72 years, was operated on early last December, and is alive and well and putting on weight, though operation revealed septic peritonitis, with escape of foul-smelling fluid and gas into the peritoneal cavity. There was a large patch of gangrene in the cæcum, which had perforated in two places; a stricture from a malignant growth, probably in the transverse colon (since it is known not to be in the descending portion), almost certainly exists; and the indication now is to deal with this if possible by excision, or, if this does not prove feasible, by lateral anastomosis and so short-circuiting the obstruction. This will allow the colostomy to close of itself.

The second case came under my care too late. The patient was a man, æt. 46 years. The conditions present were practically identical with those described in the first instance. Right colostomy was performed, and the peritoneal cavity drained, but the patient succumbed some fifteen hours later.

At the autopsy, the gangrenous cæcum was found to be associated with five or six stercoral ulcers, two of which had perforated.

A stricture barely admitting the finger-tip was found at the junction of the middle and distal thirds of the transverse colon, due to a quite small and soft annular carcinomatous growth, which seemed barely to have infiltrated the deeper layers of the bowel-wall. Proximal to the stricture the ascending and transverse colon were greatly hypertrophied and distended with fæcal accumulation.

Let me conclude these preliminary remarks by supposing that you are beginning to think that a case under your care may be one of intestinal obstruction. Under these circumstances it would be superfluous to warn you of the danger of further continuing the use of aperients in the vain hope of their relieving the "constipation"; or, again, the almost more dangerous practice of stifling the pain with morphia, and so cloaking the progress of symptoms which would otherwise guide you in the conduct of the case.

If a consideration of the history and an investigation of the abdomen (including all the well-known positions for a strangulated hernia), and of the bowel, and of any discharge therefrom, afford insufficient grounds for the exact differential diagnosis, let me urge you not to waste valuable time, but proceed at once to make the necessary arrangements for an exploratory laparotomy, since the chances of a successful issue will be greatly enhanced thereby.

CASE I. *Penetrating Wound of a Hernial Protrusion (self-inflicted); Intestine and Mesenteric Vessels Transfixed; Intestine and Mesentery Sutured; Death from Hæmorrhage, External and Internal, and Shock.*

The patient, a youth, æt. 18 years, was admitted under my care in September, 1904, covered with blood, seen to be escaping from a self-inflicted wound, about one and a half inches long, which had been made into a hernial protrusion in the right inguinal region, about the size of a large orange. This having suddenly appeared, the patient, who is stated to have been rather weak-minded, sat up on the edge of his bed and stabbed the swelling with a knife.

Small intestine had prolapsed on to the abdominal wall through the wound, which was situated at a level corresponding with the right internal abdominal ring.

At the operation, on enlarging the wound, the

swelling was found to consist of an interstitial inguinal hernia, by the side of which another loop of intestine had slipped down and out. The hernial sac did not enter the scrotum, but passed up and out towards the anterior superior iliac spine amongst the muscular planes of the abdominal wall.

Traction on the loop of bowel exposed an extensive tri-radiate gash of the mesentery, evidently the source of hæmorrhage, which had by now nearly ceased. The intestine itself was found transfixed.

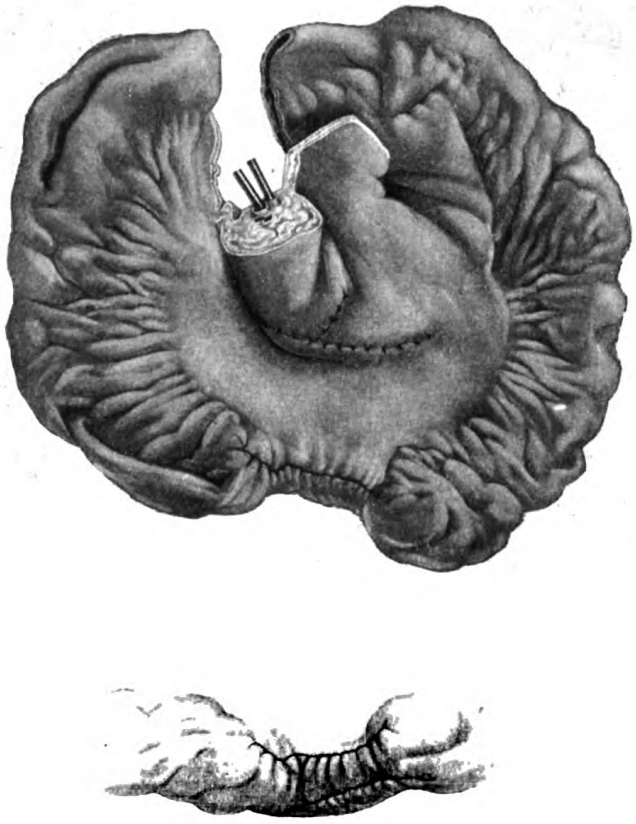

FIG. 1.—Penetrating wounds of hernial protrusion (suicidal).

The nearer incision was stitched longitudinally before the second was discovered; otherwise it would have been preferable to have sutured both transversely, or, ideally, to resect the bowel and corresponding portion of the mesentery; but this was contra-indicated, owing to the shock and the urgent need to complete the operation rapidly. Injections of strychnine, digitalis and transfusion were required at the operation.

Death occurred nine hours after admission.

At the autopsy, clots of blood were found everywhere between the coils of intestine. The source of hæmorrhage, seen to be confined to the wounded mesenteric vessels, was found to have been efficiently dealt with at the operation. Death resulted from hæmorrhage, internal and external, and from the associated shock.

The loop of bowel forming the hernial protrusion, which has been exposed for nearly five years to a mixture of formalin and spirit, is now exhibited, and is also shown in the accompanying sketch by Mr. Shiells (Fig. 1).

CASE 2. *A large Umbilical Protrusion simulating a Reducible Hernia, found to be a trabeculated sac only, containing free Ascitic Fluid (Cirrhosis of the Liver); Radical Cure; Recovery.*

The patient was a young married woman, æt. about 24 or 25 years, admitted under my care at Bulawayo on March 23rd, 1905, for an extreme degree of ascites due to cirrhosis of the liver, probably of malarial origin.

In addition, there was a very large spheroidal protrusion from the umbilicus, quite half the size

of the patient's head. It was dull on percussion and contained ascitic fluid. On palpation it appeared also to contain bowel and omentum, which, on taxis, seemed easily reducible. The umbilicus was, of course, markedly displaced downwards towards the pubes, and above it coursed numerous large veins, two being very prominent, extending on either side the middle line from xiphisternum to the protrusion below. The condition is well depicted in the photographs (Figs. 2 and 3).

FIG. 2.—Large umbilical protrusion simulating reducible hernia.

FIG. 3.

As there was marked ascites, on the following day the patient was tapped, and 21 pints, 4 oz. of fluid were removed. This was of the usual yellow tint, sp. gr. 1024, of slightly alkaline, almost neutral reaction. There was much albumen. Fehling's solution on boiling was turned a claret colour, but there was no actual reduction otherwise.

Operation.—Three days later, on March 27th, under chloroform, the very large umbilical sac was excised, and a radical cure, according to Grieg Smith's method, performed. The principle of this operation consists in removing the sac and superfluous skin, after dealing with the bowel and omentum; then the thick fibrous edges of the wound are freshened and united by buried stitches, the skin suture taking in the anterior layer of the sheaths of the recti, and is so arranged that its line is to one side of, not over, the deep sutures.

The interest of this case lies in the fact that, prior to excision, the sac was opened and found to be trabeculated throughout, so that its interior resembled a very coarse marine sponge with large intercommunicating spaces. It opened into the general peritoneal cavity by a constricted neck admitting only the index finger tip. Hence the ascitic fluid could pass in and out freely, but no hernial protrusion was possible. On taxis, the "hernia" felt as if it were being reduced, though fluid alone returned to the abdomen, whilst the presence of the very thick trabeculæ gave the impression that the sac contained bowel and omentum.

Subsequent history.—The temperature was 100·2° F. the next evening, but thereafter was normal.

Five days after the operation the patient was again tapped, 8 pints 14 oz. being drawn off.

On April 10th, a fortnight after the radical cure, with the wound healed, and after an uneventful recovery, the patient left the hospital.

The rarity of such a lesion as a granuloma, almost certainly of tuberculous nature, of the extra-peritoneal tissue of the anterior abdominal wall, seems to make the following case worth recording.

CASE 3. *Tubercle of the Extra-peritoneal Tissue of the Anterior Abdominal Wall, associated with Pleurisy and Adhesive Peritonitis (Polyorrhomenitis, or Polyserositis).*

A man, æt. about 30 to 35 years, had been ill for one month, though he had continued work up to a few days before admission under my care on December 29th, 1905, when his temperature was 98·8° F., rising later in the day to 102·2° F., the pulse and respiration rates per minute being 108 and 20.

He complained of abdominal pain and distension. There were no other definite symptoms. The abdomen was visibly distended, and lying across the epigastric region there was a large, prominent coil of bowel, apparently dilated transverse colon, thought to be fixed by peritoneal adhesions. Definite peristalsis was not observed. There was slight tenderness at McBurney's point. There was no fluid.

Chest.—At the right base behind, up to the angle of the scapula, and extending forward to the base of the right axilla, there were signs of pleurisy; elsewhere, and all over the left chest and cardiac region, the physical signs were normal. In the evening about 2 oz. of thick muco-pus, of "crushed strawberry" tint, were coughed (?) up, and this recurred two and a half hours later, but was then thought to have been vomited up.

Four hours after castor oil, there were three copious bright yellow stools. The next day the abdomen was much less painful and distended. The swelling and resistance just above the umbilicus, thought to be transverse colon, was still present, and there still remained some resistance and tenderness about McBurney's point. Elsewhere the abdominal wall was quite soft. The temperature—98° F. in the morning—rose to 101° F. by 6 p.m. The pulse failed and restoratives were required. Almost throughout the pulse was soft and dicrotic, and peritonitic in character.

For the next twenty-five days the temperature remained practically normal, but the dry pleurisy on the right side steadily extended up the back, and, finally, to the top of the axilla.

Though the bowels acted regularly with aperients, the abdominal pain became worse, especially on turning towards the right side; it was referred to the right axilla over the liver, and across the abdomen, above the umbilicus, where the patient still complained of a "tight band," especially about the mid-line of the left rectus muscle.

Median laparotomy above the umbilicus was performed on January 16th, 1906 (eighteen days after admission). On deepening the incision, a thick mass of tissue was met with, lying right across the middle line, apparently arising in the extra-peritoneal tissue, lying between the peritoneum and the deep aspect of the sheaths of the recti muscles, with which it seemed closely incorporated.

It formed an ovoid mass, having its long axis horizontal, extending in each direction to about the level of the outer borders of the sheaths of the recti, and measuring approximately 3¼ in. by 2¼ in., its depth from before back being greatest in the middle line, where it measured about ¼ in. (4 mm.), gradually fading off in depth towards its extremities.

The cut surface exposed was firm, and presented a speckled and finely nodular or granular appearance, many of the granules being of brown, almost chocolate tint, so that the surface reminded one of a "nutmeg liver." A portion was excised and sections made. Though its appearance rather suggested a fibro-sarcoma, it proved to be a mass of fibro-caseous tubercle, crowded with giant cells, with peripherally-placed nuclei. The microscopic specimens have been examined by Mr. T. W. P. Lawrence, F.R.C.S., Curator of the Pathological Museum, University College Hospital Medical School, to whose courtesy I am indebted for this expression of opinion. There is no suggestion of either gumma or sarcoma.

On laying open the peritoneal cavity, the transverse colon presented. It was distended and somewhat congested. There was no effusion of fluid and no visible tubercles. Nothing else abnormal could be detected, but the necessary manipulations probably separated some soft adhesions, judging by the subsequent course of the case.

The wound healed without incident, all the stitches being removed by the tenth day.

On January 29th there was slight abdominal pain at the seat of the "growth" incised at the operation thirteen days earlier, and a little distension, though less than on admission. Wound soundly healed.

The patient walked out of the hospital on February 2nd, 1906, with normal temperature, the bowels having acted regularly since the third day after the operation. He was warned to return if pain or distension recurred, and as he did not do so during the remaining five months of my stay in Rhodesia, one may assume, perhaps, that, during at least that period, there was no recurrence of his trouble.

Comment.—The physical signs pointed to extensive and rapidly extending right-sided pleurisy, the lung becoming adherent from base to apex whilst the patient was in hospital. The coughed-up "crushed-strawberry coloured" muco-pus, unfortunately not preserved for examination, would seem to suggest a condition of bronchiectasis, or, possibly, small vomicæ of tuberculous origin. In addition, there was the granulomatous growth, teeming with giant-cells—the sections presenting every appearance of fibro-caseous tubercle—associated with a slight degree of chronic peritonitis, indicated by tenderness over the cæcum and transverse colon, the colon at the operation, being seen to be distended and congested.

One can hardly escape the conclusion that the affections of the peritoneum and pleura were due to a common infection, probably by the tubercle bacillus. The rapid extension of the pleurisy whilst the patient was actually in the ward and the large size of the tuberculous granuloma in the extra-peritoneal tissue would seem to indicate that, following the usual course of things, the peritoneum was attacked first and the right pleura later.

"The association of similar inflammatory changes, whether acute or chronic, in two or more serous cavities," says Rolleston,* is termed "polyserositis," or, as suggested by Dr. F. Taylor, who has paid special attention to the condition, "polyorrhomenitis." "When the condition is chronic it has also been called 'Concato's disease.' Picchini (1891) argues that subacute and chronic polyorrhomenitis is practically always tuberculous, and F. Taylor,* who quotes him, accepts this in a modified way," pneumococci, staphylococci, and more rarely, streptococci, being also known as infective agents.

D'Arcy Power,† quoting two cases of his own, æt. 4 and 5½ years, the average age being between 16 and 30, says: "Resulting from a general infection, it frequently begins without assignable cause, and runs an abnormal course in weakly persons. One serous cavity is usually invaded before the others, and the peritoneum is generally attacked first, then the pleuræ, the right pleura before the left. Sometimes the pleura is attacked before the peritoneum, pericarditis following the pleurisy, especially when it has been on the left side.

"The interval between the invasion of the different serous sacs may be a few weeks, or some months.

"The prognosis is fairly good. Picchini found that in 50 cases there were 22 deaths, 21 recoveries, and 7 cases improved."

Treatment.—Where the diagnosis of tuberculosis has been made the usual general measures will be taken for the improvement of the patient's vitality with counter-irritants, such as painting with iodine, etc., in the case of the chest; in the early stages of the disease, effusions, whether pleuritic or peritonitic, will, of course, have to be dealt with as usual, pus being evacuated as it is formed. D'Arcy Power recommends mercurial inunctions in the pre-suppurative stages; and personal experience—using the ordinary blue ointment—in the case of masses of tuberculous glands *before they have actually broken down*, leads me to believe that mercury in this form really has almost as specific a power as in the disease with which it is more commonly associated.

The use of tuberculin will also, doubtless, have suggested itself to you.

April 18th, 1910.

(*To be concluded.*)

* Rolleston, "Diseases of the Peritoneum," in Osler's 'Modern Medicine,' vol. v.

* 'Brit. Med. Journ.,' vol. ii, 1900.

† 'Encycl. and Dict. of Med. and Surg.' (formerly 'Encycl. Med.'), art., "Peritoneum—Tuberculous and Other Affections."

ON SOME AFFECTIONS OF THE EYE-MUSCLES.*

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AFFECTIONS of the external eye-muscles may cause three sets of symptoms: (1) Those that affect the appearance of the patient; (2) those that affect his vision; and (3) those that produce a strain on his nervous system. The first sort of symptom —the ugly appearance caused by squint—is what brings most of the patients who suffer from it to our consulting rooms or out-patient departments. Unfortunately parents, and especially poor parents, do not realise that there is a far more important matter involved than mere appearance, and that an eye which begins to squint about the age of three, as most of them do, and which is allowed to go on squinting without any attempt to force it into use, becomes partially blind, amblyopic, as we say, from want of use. There has been a great deal of discussion as to whether it is true that amblyopia *ex* anopsia—blindness from want of use—is a fact, but it is now generally admitted that it is, and indeed the proof is convincing, for one always finds that the younger the child is at the time when the squint becomes constant the more blind does the squinting eye (in the absence of treatment) permanently remain, whereas in those cases where the squinting eye is forced into use by placing the other one artificially out of use the vision of the squinting eye is retained to a far greater extent. As has been well said by Priestley Smith, all infants are highly amblyopic at birth, *i.e.* it takes a considerable amount of experience on the part of the infant before the images formed on his retina and conveyed by means of the optic nerve to the brain have any relationship to the objects which the infant handles or sucks, and it requires that the faculty of attention should be exercised by the infant before this relationship is consciously recognised. By the age of six months this faculty of attention is only developed in relation to a comparatively few objects in the external world. In the case of all the rest the relationship between the child's consciousness and the images formed on his retinæ is a very shadowy one. If the child now develops a squint this hazy relationship between the images on the retina of his squinting eye and his consciousness becomes permanent, while in the case of the other eye the relationship develops normally as the child's experience of the external world becomes gradually wider. At the age of three years it is far wider than at the age of six months, but still not nearly so wide as at the age of six or seven years. Hence we find that an eye which develops a permanent squint at the age of six months, when its refraction is corrected at some later age—say when the child is at school—is found to be in most cases so blind that it can perhaps count the number of fingers that are held up in front of it but cannot recognise the largest letter of the ordinary test-type at twenty feet. With an eye that develops a squint at the age of three years some vision is almost always retained, but if the case is neglected till school age we shall generally find only about one quarter to one half of the vision of the non-squinting eye even with the refraction corrected. In the comparatively rare cases of squint which do not develop till the age of six or seven years the vision of the squinting eye is often nearly, if not quite as good when corrected as that of the non-squinting eyes. Amblyopia from want of use, then, is a fact, if by want of use we understand want of experience in fixing the attention on the cerebral representation of the image formed on the retina of the squinting eye. The squinting child wants to look at his toy and directs his non-squinting eye towards it. In so doing he directs his squinting eye on to some other object. His attention, however, is entirely taken up with the toy, and the image of the other object which is formed on the retina of his squinting eye, possessing no interest for the child, is entirely ignored by him. It is generally stated that the image formed by the squinting eye is suppressed. In most cases it would be truer to say that the image never gets so far as the child's consciousness. There is a block, as it were, between the last stage in the journey of the rays of light which fall on the object and the child to whom the rays of light ought to convey a meaning, so that although they get as far as his brain they do not reach himself. In some cases, however, when the consciousness has already been educated by artificial means to recognise the image in the squinting eye, and also in cases of alternating

* An address delivered before the Hampstead Medical Society.

squint when the vision of the two eyes is almost equally good from the first, there does seem to be a suppression of the image in the squinting eye in a more active sense. The curious thing is that the child hardly ever sees double. He seems to acquire a special power of, as it were, turning on the switch which awakens consciousness, either to the image formed by one eye or by the other, hardly ever to the two images at once.

So far we have been confining our attention to one of the effects of squint—defective vision in the squinting eye—and it may seem like putting the horse behind the cart to go back now to discuss its cause. My excuse is that, whereas its effect is comparatively simple, having to do with the vision of each eye separately, its cause is more complex, having to do with the faculty of *binocular* vision, a very late faculty to be evolved in the animal kingdom, the anthropoid apes being the only animals which possess it in any degree approaching to that in which the normally developed man possesses it. This being so, it is not at all surprising to find that this faculty—the being able to interpret the two separate impressions sent up by the two optic nerves, and from them to form a composite sensation which differs from that which would be produced by the message of either optic nerve acting separately—it is not surprising that this faculty, a cerebral one only recently evolved in the evolutionary history, should be feebly developed in some individuals and altogether absent in others. It is Mr. Worth, in his book on 'Squint,' who has placed our knowledge on this subject once for all on a firm basis. He has shown that in some families the absence of the sense of fusion which is the perfection of binocular vision is hereditary, that in normal children on the other hand, it is generally developed gradually between the ages of six months and six years, that the absence of this faculty is the predisposing cause of all ordinary cases of concomitant internal squint, and that in cases in which the faculty, without being totally absent, is imperfectly developed, it is capable of being trained and perfected by means of specially devised stereoscopic exercises. In the absence of this faculty it does not necessarily follow that a child will squint, but if other causes of squint exist, such as an uncorrected hypermetropia, making a continual excessive demand on the muscles of accommodation with which those of convergence are physiologically associated, or the coincidence of this condition with a debilitating illness such as measles, which makes even ordinary calls on the child's muscles of accommodation unusually trying to his nervous system—in conditions such as these existing all together the child will be fortunate if he escapes the development of an internal squint. In perfectly normal individuals, on the other hand, the presence of the faculty of binocular vision serves as an effective stimulus by means of which the two eyes are always directed towards the same object, and which is able to counteract all temptations to squint from such causes as I have just mentioned.

In the *treatment* of the convergent squint of children there are three objects to be attained: first the obvious one, which is really of very minor importance, to improve the appearance. In the old days tenotomy was performed much too recklessly, and as this was often the beginning and the end of treatment the last state of the eye was often far worse than the first, for although the eyes may have been put approximately straight at the time of operation, the gradual weakening of the stimulus to convergence in the hypermetropic child often resulted in an actual divergence of the two eyes at a subsequent period, which is certainly more disfiguring than an ordinary convergent squint. The first rule, therefore, is to correct the refraction and to be sure we have obtained the full effect of convex glasses in correcting the squint before we think of operating. When hypermetropia exists the squint is almost always partially corrected by the appropriate glasses, and in a few cases it is completely so, proving that the connection between accommodation and convergence is an important factor in these cases. When the correcting glasses have been worn for six months it will be time enough to think about improving the appearance by operation, and when that time comes it will often be better to strengthen the muscle that is too weak by an advancement rather than to weaken the muscle that is too strong by a tenotomy.

But after all, at any rate during childhood, the appearance is a matter of minor importance; what is far more important is to prevent the sight of the squinting eye from deteriorating, and in attaining this object it is impossible to begin too soon—indeed, the younger the child is when he begins to squint the more essential is it for treatment to be

prompt. There is one and only one way of effecting this object, and that is to force the child to use the squinting eye by preventing him from using the other one. In cases of alternating squint where either eye is used indifferently from the first no artificial means of effecting this object will be necessary, but in all other cases the good eye should be put out of use by an opaque glass or other means. In some cases it will be sufficient to keep it under the influence of atropine. Of course, when the squint has been neglected too long even this treatment will not be any good, but in all cases, except when the vision of the squinting eye is so low as not to be able to count fingers at six feet, it should be tried. In cases where this treatment is partially successful it can be usefully continued at a later stage by the use of cards devised by Mr. Alison Bradburne, of Manchester, by which the bad eye is gradually educated by the good one to recognise smaller and smaller letters.

The third thing to be aimed at in the treatment of convergent squint is the cultivation of the faculty of binocular vision. The most elementary development of this faculty may be tested by a litttle instrument devised by Mr. Priestley Smith, which shows whether or not the patient has *simultaneous* vision of the two images in his two retinæ. If he has it may or may not be worth while to attempt to train his fusion faculty, and for this purpose Mr. Worth's "amblyoscope" is the best instrument that I know of. In a favourable case the exercises with this instrument can be prescribed for home use, and when the fusion faculty is present it can be trained until the patient fuses the two images so perfectly as to get the true sense of perspective. When a case has progressed as far as this it may be considered as a complete cure, for the sense of perspective, once acquired, is never lost.

Now a few words about the other main division of cases of squint—paralytic squint. These are divided from the concomitant variety by one essential distinction. Whereas in the latter the movements of each eye separately are entirely normal in every direction, in the case of paralytic squint they are limited in the direction in which the paralysed muscle normally turns the eye. A less fundamental distinction, but at the same time a very convenient one, is that in concomitant squint there is hardly ever diplopia. In the paralytic variety, on the other hand, diplopia is always the symptom which brings the patient to consult us. These patients, as a rule, are adults. The relation between the images formed on the two retinæ and their consciousness is so intimate that any want of correspondence between the two images at once produces double sight. This, however, is not such a fundamental distinction as the first, because diplopia is not unknown in cases of concomitant squint in children which begin unusually late. The only cases in which there can be any difficulty in the diagnosis, of course, are those of paresis of the external rectus, and here the secondary deviation of the non-squinting eye when shaded, while the squinting one is directed to look in the direction in which movement is difficult, is enough to distinguish these cases from those of concomitant squint, in which the excursions of the associated muscles of the two eyes are always equal.

To distinguish between the paralyses of the various eye-muscles requires a certain amount of care and an exact knowledge of the various actions of the muscles in question. The only real difficulty occurs in distinguishing between the vertically moving muscles, *i. e.* between the superior rectus and inferior oblique, both of which move the eye upwards; or, on the other hand, between the inferior rectus and superior oblique, both of which move the eye downwards. It will be enough now to mention the most common cases of paralytic squint, those, namely, due to lesions of the third, fourth, and sixth cranial nerves respectively. A lesion of the third causes paralysis of nearly all the external as well as the internal muscles of accommodation and pupil contraction, and also the levator palpebræ. A lesion of the trunk may produce all these effects, but it is more common to see cases in which the internal muscles escape and some of the external muscles also, and this points to a lesion affecting, not the trunk of the nerve, but part of the nucleus, which, you will remember, is a very large one occupying a large space in the mid-brain. In a case of complete paralysis of the third the only muscles which escape are the external rectus, supplied by the sixth nerve, and the superior oblique, supplied by the fourth. Cases of paresis of the superior oblique are sometimes difficult to be sure about, as the defect in movement may be so slight as to entirely escape observation, and the only means of diagnosis is the location of the double images by the patient himself. I will not

attempt to go into the details of this diagnosis just now, but will just mention the two most common causes of ocular paralysis, which are—(1) traumatism, and (2) syphilitic or para-syphilitic affections of the nervous system, including tabes dorsalis.

As an instance of traumatic paresis, E. R—, a woman, æt. 56 years, came to the Western Ophthalmic Hospital on September 4th, 1909, complaining of double vision. There was no obvious defect of movement, but diplopia existed and was increased on looking downwards. The muscle affected, therefore, was one of the depressors of either the right eye or the left. The *lower* of the two images was that corresponding to the right eye, therefore the eye affected was the right one. The vertical separation between the two images was greater on looking down and to her *left* than on looking down and to her right, *i. e.* it was most obvious when the direction of the gaze was most in a line with the tendon, not of the inferior rectus but of the superior oblique, or rather that portion of the tendon between the pulley and the attachment to the eyeball. Therefore the muscle affected was the right superior oblique. The diplopia had been present for nine weeks since a fall on the head which made her unconscious. There was the scar of a scalp wound in the right parietal region which had been treated at St. Mary's Hospital after the accident. The cause of the diplopia was probably a slight hæmorrhage into the right orbit which interfered with the action of the superior oblique tendon.

As to treatment, the obvious and only measure to relieve the annoyance of diplopia is to shade one eye—it does not matter which if the vision of both is equal. In syphilitic cases, of course, the remedy is also obvious. In tabes the diplopia is generally transient in itself, but liable to recur. In some old cases no treatment seems to be any good. In one of these, in which there had been a paresis of the right superior oblique lasting twelve years, I did an advancement of the right inferior rectus with the effect of making the diplopia less annoying to the patient without altogether curing it.

I said at the beginning of my paper that affections of the eye-muscles are attended by three sets of symptoms, and as yet I have confined my remarks to some aspects of the first two—those which have to do with appearance and those which have to do with vision. Before concluding my paper I must briefly touch on the third set of symptoms—those which indicate a strain on the patient's nervous system. These symptoms are characteristic of quite a different class of cases from those with which I have already dealt—cases, namely, in which binocular vision is perfect and there is no squint, paralytic or otherwise, but in which the condition of the muscles, when the two eyes are normally functioning, is not one of equilibrium but one of strain. In a case in which the muscular equilibrium is perfect, when the two eyes are directed to any object, whether distant or near, and a shade is placed before one eye, thus preventing it from seeing the object, its position remains unchanged. When, on the other hand, the equilibrium is imperfect, the position of the shaded eye does not remain unchanged. It moves outwards or inwards, or less commonly, upwards or downwards. The same effect is produced by altering the nature of the image due to a spot of light formed in one eye by means of a Maddox rod, which may be used in combination with prisms as a test for the amount of defective muscular balance when any exists. The most common instance of such defect is weakness of convergence, a condition which, in patients subject to it, varies very much, depending largely on the state of the general health. Leaving these cases out of account there may be want of balance either between the laterally moving muscles or the vertically moving ones. In the case of the laterally moving muscles there may be, and often is, a considerable want of balance without any symptoms whatever. The external and internal recti are so comparatively strong that they are generally quite able to overcome a slight tendency either towards convergence or divergence without producing any nervous symptoms. In America, indeed, there are some ophthalmic surgeons who make a habit of prescribing for these slight defects even without the existence of any symptoms referable to them, but in this country, at any rate in my experience, the cases where it is advisable to prescribe prisms with bases either in or out are not very frequent. In the case of the vertically acting muscles the case is otherwise. The want of vertical balance is far less common, but when it does exist it is far more liable to produce annoying symptoms such as headache or general malaise. This is not surprising, because the strength of a vertically placed prism that can be overcome by a normal individual without producing diplopia is only from one third to one half the strength of a prism placed with the base in, and a much smaller proportion of the strength of a prism placed base out. The moral is that in want of balance between the vertically moving muscles of the two eyes, when there are any symptoms of nervous strain, it is very good practice to prescribe a vertical prism calculated to nearly counteract the error that exists.

April 18th, 1910.

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CONTENTS.

PAGE

*Treatment of Colitis. By HERBERT P. HAWKINS, M.D., F.R.C.P. 33

*Interesting Cases of Abdominal Surgery, including some Rare Forms of Intestinal Obstruction. By HENRY CURTIS, B.S., F.R.C.S. (*concluded*) ... 42

*A Case of Myomectomy During Pregnancy. By FRANK E. TAYLOR, M.A., M.D., M.Sc., F.R.C.S., M.R.C.P. ... 47

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TREATMENT OF COLITIS.*

By HERBERT P. HAWKINS, M.D. F.R.C.P.,
Physician to St. Thomas's Hospital.

THERE are many varieties of colitis with a wide range in causation and severity. The varieties are mostly unnamed and hard to separate, and in very few of them has a specific nature been established.

If a discussion of the treatment of such a wide disease is to be of the greatest service we must agree upon some sort of classification. I think we are safe in recognising four large simple groups.

(a) In the first place I would dismiss the colitis due to protozoa as not being within the first-hand knowledge of most of us. It may be noted, however, that Professor Saundby has reported a case of amœbic dysentery arising in this country. Amœbic dysentery, like bacillary dysentery, may lead to intractable ulceration of the colon, the perpetuation of the lesions and the refusal to heal being probably due to secondary bacterial infections. It is in this state that we often see patients admitted into our hospitals. I think they require the same treatment as any other form of ulcerative colitis. The state is often incurable, but it is compatible with many years of life if an efficient colostomy is established.

(b) In the second place I would summarily dispose of a "toxic" colitis as being unimportant from our point of view of treatment. It includes a consideration of a bacterial poison. But here the organism is not contained within the bowel, but is situated elsewhere (as, for example, in all septicæmic conditions), producing its morbid effect during the excretion of its toxin by the colon. Here also is placed the colitis of mercury-poisoning and of mineral acids, though the mode of action is probably different in the two cases, the former acting indirectly by excretion, the latter by local

* Contributed to the Medical Society of London.

contact. As an example of the excretory function of the colon may be cited the fact that colitis can be produced by the inunction or injection of mercury as well as by oral administration. Another instance under this head is the colitis of renal disease, pneumonia, and many severe diseases, the colitis being probably in all cases of excretory origin.

(c) In the third place we have the large group of bacterial colitis, in which the bulk of our cases, and certainly the most severe forms, must be placed.

In this group the attacking organism is contained within the intestine. It may be one of the normal and ordinarily peaceful flora of the bowel, but in the graver cases it is probably one of the "wild races," which are often introduced with food and drink (especially by fly-contamination), from unclean mouths and teeth, and possibly from swallowed nasal and bronchial secretion.

There are many forms of bacterial colitis, and it is here that the need of some classification is most felt. One pines for some name for each form, some name that will carry with it a definite idea as to its causation and as to the routine treatment most likely to be of service. No classification on a basis of symptoms is possible. The points of difference are too few. But no one can doubt that we shall eventually possess a true bacteriological classification. All we can do now is to divide up the cases we have seen on the basis mainly of severity, duration, and extent of lesion. We can, of course, enumerate various bacteria which from purity of growth and from *à priori* knowledge may be regarded as the probable cause in certain cases. But, except in the cases of typhoid, para-typhoid, and tuberculous infections, which can be recognised by clinical experience and by serum-reactions, we are not yet able to fit the organism to individual cases of colitis with any degree of precision.

We can, however, rough out certain classes, but it will be realised that these classes are formed by "looking back" on the notes of cases, and not by "immediate bedside recognition." Further, it is obvious that this is no kind of classification, inasmuch as the same organism (*e.g. B. Flexner*) can be responsible for cases in more than one class, and that these classes, therefore, often only represent stages or degrees of one disease.

For the purpose of treatment the broad distinction between a catarrhal non-necrotic colitis and a necrotic ulcerative form is of great importance. The one form can be regarded without hurry; in the other immediate action is necessary. But even this distinction cannot be drawn with a hard line. A patient who comes before us presenting advanced ulceration of the colon may have a long history of previous attacks of comparatively mild diarrhœa, so that we realise that a catarrhal form may pass into a later stage of ulceration and yet presumably be due to the same organism. This same variation in the severity of symptoms is seen in bacillary dysentery of the tropics. Moreover, in many cases of colitis, which are by no means severe and which are mainly catarrhal, it is probable that superficial ulceration and abrasions occur which do not proceed beyond the point of speedy repair, and we may have to wait till the end of the illness before we can say to which class a given case belongs.

(A) *Catarrhal Colitis.*

In mere frequency of occurrence a simple acute catarrhal colitis exceeds all other forms of colitis. It is especially common in the summer and autumn. Those who have previously suffered from dysentery are liable to such attacks, which are readily provoked by chill or errors of diet. The symptoms and course require no description. It is of short duration, easily remedied, and at the moment produces no anxiety. It is occasionally followed by appendicular pain and tenderness, or by actual appendicitis. Many such cases are called "enteritis" as being an inflammation of the small intestine rather than the colon. Though the small intestine is doubtless infected, I believe the actual cause of the diarrhœa is the condition of the colon. In fact I doubt if a violent diarrhœa ever arises from a pure infection of the small intestine. Even in such conditions as typhoid, paratyphoid, and tuberculous infections I doubt if diarrhœa is produced, unless catarrh of the colon is present, and in all three instances such a catarrh is of common occurrence.

A similar catarrh is present in those cases of carcinoma of the colon, especially the pelvic colon, which come before us with a history of diarrhœa. It is exceedingly easy to make a mistake in such cases, and the use of the sigmoidoscope is essential,

The same mistake is possible in cases of general polyposis of the colon, so-called "colitis polyposa."

As regards the bacteriology of a simple acute catarrhal colitis, in this country Morgan's bacillus and *B. enteritidis* are prominently before us. In America *B. Flexner*, or *B. Shiga*, or both, seem to play the chief part, especially in the ileo-colitis of children. It is just possible that putrefactive anaërobes, such as *B. aërogenes capsulatus*, may be engaged. But in this form, as in many others, the question remains unanswered as to the possibility of *B. coli* gaining harmful properties through some alteration in its own power of growth or some lessening of the normal resistance of the intestinal mucosa.

(B) *Necrotic Colitis.*

(1) *Diphtheritic or croupous colitis.*—This form I should like to dismiss at once. Shreds of membrane consisting of necrosed mucosa and coagulated fibrinous exudation may sometimes be seen on a small scale in cases of ulcerative colitis, which have proved fatal in an early stage. But the pure condition in which one or more localised patches are suddenly developed in the colon without other disease has no importance from the therapeutic point of view, owing to its rarity and the impossibility of diagnosis. I have only seen two cases. One was a terminal infection in a case of splenic anæmia. The majority of cases are probably terminal infections. In the other case, in which the diphtheritic process was limited to a few inches of the left colon, the symptoms were those of acute obstruction and laparotomy was performed. I know of no observations as to the bacteriology of this condition.

(2) *Secondary or associated colitis.*—This also is an unimportant group. Most cases coming under this head are really of toxic rather than infective origin, but occasionally a true bacterial colitis occurs in association with disease of a similar nature elsewhere. As an example may be taken the case of a child who had been in St. Thomas's Hospital for sixteen days suffering from basal meningitis. Profuse diarrhœa with passage of blood then began, and continued till death fifteen days later. Post mortem there was found purulent basal meningitis, and the colon was closely set with shallow ulcers. Pure cultures of pneumococcus were obtained from the meningeal pus and from the splenic blood, and numerous similar cocci were found in the exudate on the surviving mucosa of the colon.

(3) *Ulcerative colitis* is a matter, concerning the treatment of which all available experience is welcome. The mortality is great, probably not less than 70 to 80 per cent. It is certain that bacteriologically there are several different forms, and if the use of a vaccine or serum should eventually become a routine treatment, it is necessary to distinguish between them.

(*a*) *British dysentery.*—Of the family group of *B. Shiga* and *B. Flexner* one or other may be accepted as the main cause of asylum dysentery. It may be taken as certain that non-institutional cases have a similar origin, and that they are identical clinically and bacteriologically with bacillary dysentery of the tropics. It is possible that the small epidemics which still occur in this country are of the same nature. In the summer of 1901 between 170 and 180 persons were so attacked at Aldershot, of whom 38 died.

An example may be given :

Man, æt. 48 years, had, in 1905, a severe attack of diarrhœa with griping pain. In 1907 he had a second attack, in which he passed blood and mucus, and in 1908 a similar attack of shorter duration. All three attacks occurred in July or August, the season of flies. He had never been out of England. On July 12th, 1909, his fourth attack began with diarrhœa, griping pain, tenesmus, and passage of red blood and mucus. He was at once given 20 c.c. of Shiga-Kruse serum, with calomel and opium. He left hospital well in seven weeks, and in January, 1910, remains well. It is quite possible that he will have another attack this summer. From a high rectal scraping *B. Flexner* was obtained. The patient's serum agglutinated this bacillus up to a dilution of 1 in 100. A rabbit was immunised with the bacillus isolated. The rabbit's serum gave no agglutination reaction with the bacillus of Morgan or *B. Shiga*, but a reaction was obtained with this bacillus and with *B. Flexner* up to a dilution of 1 in 1000.

Another example is that of—

Woman, æt. 41 years. Sudden diarrhœa ; stools every fifteen minutes at first, with straining and genuine tenesmus and pain on defæcation, most stools containing red blood. Tongue red and dry. Temperature normal. Treated with bismuth, and

opium and creolin irrigation; quickly improved, and was discharged apparently cured on the fortieth day of illness; subsequent history unknown. Her serum (1 in 100) agglutinated Shiga's bacillus at once. It is highly probable that this was a case of bacillary dysentery.

Probably of the same nature is the case of—

Man, æt. 59 years. Sudden onset of diarrhœa with cramp in legs and abdominal pain, mostly in right iliac fossa. Vomited three times. Stools five or six a day— much blood and mucus. Slight fever—99°–101° F. Hiccough. Tongue became red, glazed, and dry. Pulse 132; failing rapidly. Death on twenty-third day. Post-mortem: Colon ulcerated from end to end; ulcers ovoid, with swollen edges, separated by strands of sodden mucosa; sometimes confluent over large tracts, mostly extending down to muscular coats, a few to peritoneum. No disease elsewhere. From splenic blood after death *Bacillus coli* and *Bacillus proteus* grown. No examination of a rectal scraping was made.

As regards the frequency of occurrence of this true bacillary dysentery in this country we are in doubt. But it must be remembered that the common failure to find the dysenteric organism in such cases may be explained by the fact that the fæces and not a rectal scraping have been examined. Moreover, it is highly probable that, in the advanced chronic stages in which the examination is often made for the first time, the disease is being perpetuated by secondary infections with other organisms, such as *B. coli*, *B. pyocyaneus*, and pyogenic cocci.

(*b*) It is highly probable that an ulcerative colitis may be caused by organisms of the *coli*, para-colon, and para-typhoid group, and this form may eventually be differentiated and may be further split up. As regards symptoms and the high rate of mortality, it does not differ materially from true dysentery.

An example may be quoted:

Man, æt. 23 years. In June, 1908, he had his first attack. Sudden onset of violent diarrhœa; stools up to ten a day, some containing bright red blood, much slime; severe griping across lower abdomen. Under treatment this subsided. In three weeks he was beginning to improve, and in six weeks he was apparently well again, and his stools became normal and formed. On December 11th a second attack of the same kind began. Profuse diarrhœa with blood and mucus; great rectal pain as well as general griping; vomiting, fever. This soon proved to be more severe than the first attack, and he began to lose weight. In January, 1909, his rectum was very red, granular, and moist, and it bled at the least touch.

In the next fortnight the patient went rapidly downhill, with great wasting, severe pain, raw tongue with aphthæ, and parched throat. Right colostomy was therefore performed. In spite of the diversion of fæces and irrigation, the patient died nine weeks after the commencement of his second attack. Only a small part of the colon could be obtained for examination, namely, the transverse colon and splenic flexure. Over the whole of this the muscular layer was exposed, only a few polypoidal muscular fragments of mucous and submucous layers remaining. Such fragments showed: (1) Infiltration of mucosa and submucosa, and, to a less extent, in the other coats with small and large mononuclear cells, some plasma-cells, a few eosinophiles, no polynuclear cells or basophiles; (2) thrombosis of capillaries and small vessels; (3) hæmorrhagic œdema of muscular coats; and (4) bacilli, short and fat, long and thin, throughout the whole intestinal wall, an infective condition which accounts for the toxæmia and the failure of treatment. The bacteriology is detailed in the 'British Medical Journal,' March 27th, 1909.

(*c*) A pneumococcal ulcerative colitis is known to occur. A case is recorded in the 'Proceedings of the Royal Society,' vol. iii, No. 2, which was attended with high fever and severe hæmorrhage. The good effects of running iced water through the colon from an appendicostomy opening were very striking.

(*d*) A streptococcal ulcerative colitis is also a known occurrence. I think the following case is probably an instance:

Man, æt. 43 years, was seized on December 26th, 1907, with severe abdominal pain and diarrhœa. The latter, however, was never very urgent, the stools being seldom more than three or four a day, and there was no certain history of the passage of blood. On January 28th, 1908, when I saw him, he was greatly wasted and moribund; he presented signs of pleurisy on the right side and an enlarged liver, with 38,000 leucocytes per c.mm. The colon was found to be acutely inflamed from

end to end; it was closely set with shallow ulcers, which did not seem to penetrate the submucosa; here and there were visible points of pus or miliary abscesses in the substance of the mucosa. The portal vein was distended with clots and reddish pus. There was one large abscess in the liver and several smaller ones. From the colon and from the hepatic pus only *B. coli* was grown; but a photograph of a section of the colon shows that the bowel wall was infiltrated with cocci arranged in chains. When such points of pus are found in the mucosa in tropical bacillary dysentery they are generally found to contain streptococci.

(*e*) *An ulcerative colitis* occurs in which hæmorrhage from the colon, with little or no diarrhœa, may so predominate that it may be roughly termed "hæmorrhagic colitis." The mortality is very high. After death the colon is ulcerated, as in the more common form, and it presents no differentiating feature.

An example may be quoted:

Schoolboy, æt. 18 years. In November, 1903, he noticed a little blood in motions, but there was no pain and no diarrhœa. All through 1904 he passed a little blood from time to time. In December, 1904, there was a sudden extension of infection upwards; he was seized with severe pain, straining, and tenesmus, with frequent (hourly at first) passage of liquid stools of blood and mucus. From this time he was confined to bed. During the first six months of 1905 there was almost daily hæmorrhage, with wasting and profound anæmia. In July, 1905, right colostomy was performed. He gradually improved simply as a result of the non-use of the colon. Irrigation was tried at times, but never for long and never thoroughly. In January, 1909, he has taken his degree at a university; he can play tennis, and bicycle for forty miles, and goes to dances; he looks and feels in perfect health; the artificial anus has acted continuously and well. But his disease remains uncured. About twice a day he passes a little blood-stained mucus.

I know of no certain bacteriological evidence which would enable us to place this kind of case, but there is no good reason to suppose that it is bacteriologically a separate form of disease.

(*f*) *An ulcerative colitis* occurs, which in its final stages and in the post-mortem appearances differs in no way from the first two forms, but differs greatly from them in the manner of onset or rather in its early history.

A diarrhœa may begin suddenly with no great severity and may soon subside under simple medicinal treatment. It is a simple catarrhal colitis, such as is common enough in the summer months. But the bowel never resumes its former regularity of action. Occasionally aperients are necessary. Often there are loose actions for a few days. Up to this point it may be regarded as a chronic catarrhal colitis. But a second more severe attack occurs, and perhaps a trace of blood may be reported. Blood may be present in considerable amount in later stages, but it has not the same immediate serious import as in the forms previously described. The story of such repeated attacks, often with hæmorrhage, with little or no impairment of health, except during the height of an attack, may run on for years. In fact the long duration without much failure of health and without wasting is as characteristic of this form as is the insidious onset. It is as if we had to deal with a purely local condition in the colon devoid of any toxic quality. Of many such patients whom I have seen I do not know the ending. I know, however, from post-mortem evidence that this condition may end in intractable ulceration. I have known a patient to have such attacks spread over sixteen years. At the end of this time there was so much evidence of ulceration and so little prospect of recovery under any other treatment that right colostomy was performed and recovery was finally reached. In another case for four or five years the attacks occurred with such regularity about the month of June that the patient convinced herself that it was related to hay fever.

I know nothing of the bacteriology of this kind of case. But it is possible that it belongs to the second class, differing only in the long duration and the initial mildness. The importance of it from the therapeutic point of view lies in the deceptive nature of the early stages.

The treatment of an acute catarrhal colitis presents no difficulty, and little room for difference of opinion. Only it must be remembered that in a very minute proportion of such cases it may prove to be the early stage of more serious disease. As regards drugs, I suppose that in theory the use of an initial dose of castor oil is probably correct. But in actual practice the man who quickly relieves

the discomfort and diarrhœa with bismuth and opium is most likely to be popular.

If, as time runs on, we become aware from the continuance of the trouble and from the occasional appearance of mucus and of traces of blood that we are dealing with some more serious infection than a simple catarrhal colitis, more active measures are necessary. Still more are they necessary in such cases as I have mentioned, in which the diarrhœa is violent and hæmorrhagic from the first and the constitutional disturbance is severe. In considering the treatment, I should like to fix attention on the caution with which an apparent cure should be regarded. These cases that I mention, including many of great initial severity, may be taken to show cures under the simplest treatment, but they have eventually relapsed and proved fatal.

(*a*) *Specific treatment.*—As regards the bacteriology of all forms of colitis this may be said. It is unlikely that except in the earliest stage any one of these forms is entirely due to a pure and simple infection by any one variety of micro-organism. The intestine differs from most other parts of the body in being already the holder of bacteria, some of which, though they are innocuous in health, can certainly assume pathogenic power under altered conditions. Upon the receipt of a new organism from without (*e. g.* such a well-defined specimen as *B. Flexner*) the first result of an ensuing catarrh is a modification of conditions, especially of food-supply, which can conceivably favour an abnormal growth of pre-existing inhabitants. Under the whole subject lies the question of the mutual effect of one organism upon another, of an enhancing effect (as of *B. enteritidis* on *B. typhosus*), and of an antagonistic effect (as perhaps with *B. coli* and *B. pyocyaneus*).

There is much to be learnt about the pathogenic properties of such organisms as *B. coli*, *coli*-form bacilli and *B. pyocyaneus* under altered conditions. *B. pyocyaneus* is commonly found in ulcerative colitis of all kinds, both in the fæces and in the scrapings from the bowel wall, both cæcal and rectal. In some epidemics it has clearly been the predominant organism. I have heard of a case in which great improvement or cure was attributed to a *pyocyaneus* vaccine.

Though these considerations point to a mixed infection it is not necessarily a bar to specific bacterial treatment, but the efficacy of such treatment may be found to vary with the earliness of the stage at which it is used. By the use of his serum in Japanese dysentery Shiga has reduced the mortality from 22–26 per cent. to 9–12 per cent. I have used the Shiga-Kruse serum of the Lister Institute in a great many cases without being able to point to any clear result, except perhaps in the case already mentioned, and in another case equally acute and in an early stage. In most of my cases, however, the disease was far advanced, and much other treatment was employed at the same time. Still, in anything like an acute case with early appearance of blood I should always give one or two doses of this serum. In my experience the use of a vaccine, either of a stock *coli* vaccine or of a vaccine prepared from the patient's *coli*-form bacilli, has produced no obvious result, but my cases have mostly been in an advanced stage of disease. Earliness of treatment is essential, and I am not without hope. Hale White and Eyre have reported three instances of apparent cure in severe cases by vaccine treatment ('Proc. Roy. Soc. Med.,' vol. ii, No. 8).

(*b*) *Irrigation of the colon.*—Disinfection of the bowel is out of the question. It is probable, however, that much can be done towards cleansing. In any controversy as to the relative merits of rectal irrigation and flushing from an appendicostomy opening I should certainly vote in favour of the latter. In my opinion it has everything to recommend it, especially ease, thoroughness, and absence of discomfort. Moreover, after a time the patient can do his own flushing. One of my patients has irrigated herself nearly every day for eighteen months. She keeps in fair health, but the passage of blood from time to time shows that ulceration is still present, and colostomy (which I should prefer in her case) cannot be carried out at present.

There is another consideration which, I think, tends in favour of flushing *per* appendix. It is, of course, understood that various bacteria may provoke an inflammatory reaction in the colon by the mere contact of their life-products with the mucosa. We have, then, a locally acting cause, more or less amenable to local treatment. Probably the less serious short-lived catarrhal inflammations are produced in this way. But there is considerable evidence (Flexner and Sweet) to show that

the more serious and deeper lesions of the mucosa, submucosa, and muscular layers of the colon are not produced by any such local direct bacterial action from within outwards, but that they arise in the very substance of the colon wall during the excretion of the toxin, which is being produced by bacteria situated, it may be, elsewhere in the bowel. The "undermining" of the mucosa, often seen in ulcerative colitis, is thus intelligible. The bacterial action is then indirect. We are driven to look upon the colon as an excretory organ, ranking after the kidney and liver, but, like those organs, liable to suffer during the performance of this function.

This indirect elimination process of a toxin (which can be proved experimentally with Shiga toxin), as opposed to a direct local effect of bacteria on the mucosa, is supported by the fact that in man the small intestine in bacillary dysentery is just as much infected as the colon, and yet it rarely shows more than a catarrhal reaction, and the naked-eye lesions are almost confined to the colon. It is possible that the good effect of magnesium sulphate in bacillary dysentery may be explained by its flushing and cleansing effect on the small intestine, regardless of the more obvious lesions in the colon. At any rate this conclusion may be drawn from experimental work, which, I think, is in agreement with clinical experience, viz. that it is easy and unwise to under-estimate the "extent" of the bowel which is infected. The localisation of pain in the left iliac fossa and sigmoidoscopic observation of hyperæmia and ulceration of the rectal mucosa do not necessarily justify us in believing that the disease is limited to the lower end of the colon, and they do not preclude the existence of disease at the flexures, or in the cæcum. It is generally better, therefore, to attack the disease from above than below, from the appendix rather than from the rectum.

As regards an irrigating solution, I have not used many out of the large number which are at our disposal. I rather doubt if disinfectants are any better than cleansing fluids. Normal saline and boracic acid solution seem to me all that is necessary. Methylene blue, 1–5000, I have often used. In severe ulcerative states, especially for irrigation through a colostomy, I use silver nitrate and sometimes hydrogen peroxide. If hæmorrhage is severe adrenalin may be used.

(*c*) *Colostomy versus appendicostomy.*—I feel sure, however, that in some advanced cases right colostomy is imperative for the saving of life. The choice between the two forms of treatment may be very difficult, the difficulty lying in the determination of the extent and depth of the ulceration. Appendix flushing seems to me to be certainly the right form of treatment in all chronic catarrhal states, even if a little blood has been passed from time to time. But when it can be realised from the amount of hæmorrhage, from the extent of colon tenderness, from the wasting, and from the appearance and general state of the patient that he is gravely ill, and that the greater part of the colon is deeply ulcerated, then I am equally sure that colostomy, which affords rest to the part, coupled with efficient cleansing by irrigation, is the only chance of saving life. This step I recommend, though I know well that in the majority of such cases the patient must be content with an artificial anus for the rest of his days. The chance of closure of the opening and regaining the use of a colon, which has been disused for many months or a year, even if healing should have occurred, is small owing to the progressive contraction of the colon. Similarly our attempts at St. Thomas's at ileo-sigmoidostomy in such cases have for the same reason failed. I have had only one permanent cure after colostomy and subsequent closure of the opening. The final closure in this case was effected seven months after colostomy had been performed.

(*d*) I would ask the question whether in such cases as I have described in Class (*f*) which are characterised by an insidious onset, by a long course and by the absence of toxæmic symptoms, mere removal of the appendix has been known to produce any result. I have seen some cases of this kind in which pain and even tenderness over the appendix, with a rise of temperature, have occurred from time to time associated with the diarrhœa, so that it appeared probable that the appendix as well as the colon was infected. Is there any evidence to show that the appendix may be the master-seat of the infection and that its removal may be followed by subsidence of the colon trouble? In two cases I am inclined to believe that this result did follow removal of the appendix. In two other similar cases the appendix was removed, but it is not yet time to speak confidently as to the result.

(*e*) *Lactic acid milk.*—I have used sour milk in a large number of cases. In one case of advanced ulcerative colitis, milk in which the Bulgarian bacillus had been incubated for twelve hours was injected daily for a long time through an appendicostomy opening. In a similar case it was used as a rectal irrigation. I have given dairy-made and home-made tabloid-produced sour milk in many cases of the chronic catarrhal colitis which I have mentioned, in which the appearance of blood from time to time indicates that some degree of ulceration has occurred, and I have used similar productions in many cases of mucous colitis. In no case have I seen any result whatever.

Of course it is quite possible that with the exception of the two advanced cases mentioned above no patient really received the true Bulgarian bacillus. On the other hand it may certainly be said that the good results, which I heard of, but never saw, in the early days of the sour milk boom, must have been psychical, as few if any of the tabloids then in use contained any kind of lactic acid bacillus. However, one sees even now statements of improvement under its use as having occurred, both in mucous colitis and in hæmorrhagic colitis, though other treatment, especially by vaccines, seems generally to have been employed at the same time. But I have never seen any proof of the common statement that the Bulgarian bacillus has in the intestine an inhibitory effect on the growth either of *B. coli* and *coli*-form organisms or of anaërobic putrefactive organisms. The rational use of sour milk stands or falls with the proof of such an influence. However, if well made it is a wholesome and rather pleasant drink, and I order it freely all round.

(*f*) *Medicinal.*—In nearly all forms of colitis I have great faith in the use of calomel. It is truly remarkable how well the drug is borne. It can be used for several months continuously if necessary, in small doses of gr. $\frac{1}{6}$, $\frac{1}{4}$, or $\frac{1}{2}$, two or three times a day. I generally combine with it small doses of opium, varying the proportion of the two drugs according to results. It is probable that the calomel is itself excreted into the colon, as is the toxin of *Bacillus Shiga*, and perhaps of other organisms.

(*g*) *Kataphoresis.*—I have but little experience of this method of treatment. The cases in which I have employed it were not likely to improve. But much information is contained in a paper by Wallis and Bruce ('Proc. Roy. Soc. Med.,' vol. viii).

(**d**) Finally, there is a class in which nervous phenomena, either local (secretory, motor, or sensory) or general, or both local and general, are a prominent feature of the disease. It is a question whether these nervous phenomena are primary, or secondary, or coincident with the colon affection. In many cases I am convinced that they are primary or precede the local trouble. At any rate they are an essential part of the condition, they are always present in some degree, and they must be allowed to modify the treatment. The local phenomena indicate disturbance or perversion of function, and consist of spasm pain and a vast secretion of what we may call mucus, though Leathes has shown that large casts are chemically not true mucin, but are more nearly allied to chitin. The general phenomena mainly coincide with neurasthenia, by which term is meant a quick fatigue, little reserve, and slow repair. The term "neurasthenia" does not, however, cover the whole ground. There is often added that state which greatly increases the difficulty of treatment—viz. a tendency to exaggerate small symptoms, a fanciful-ness combined with hopeless obstinacy, and a weakening of the normal healthy desire to get well. Such a mental state is never present in the purely bacterial forms of colitis already described, and it stands out in strong contrast. Moreover, in this group alone the question of sex comes in, there being a great preponderance of cases in the female.

I believe there are two kinds of cases which come under this head.

(*a*) *Mucous colic.*—The passage of mucus in masses or casts, preceded and attended by severe pain and constipation, with abundant evidence of tight spasm of the colon, generally on the left side, in attacks with long or short intervals of fair health, which are marred only by constipation, forms a definite picture of a condition easily recognised. There is no form of colon-disease so sharply defined.

I think we should use the term "colic" here advisedly and refuse the word "colitis." There is no trace of inflammation. The only organised elements in the dejecta are epithelial cells torn off and entangled in the mucus, though occasionally

a few streaks of blood may be added at the height of the paroxysm during which the mucus is extruded. I believe that what fluid is passed with the mucus is a pure transudation, which, like the mucus, may be taken as an evidence of secretory disturbance.

I am confident that such pure cases occur, in which there is no visible evidence of inflammation. Post-mortem observations have certified that absence of any visible lesion. The condition has a striking resemblance in all points to true asthma. The two conditions may occur and alternate in the same patient, and I think they tend to appear in the same family-stock.

The nervous element, local and general, is very prominent, as it is in asthma. But the known fact of the localisation of the mucus-production to a small area of the colon may well mean that some local irritant, bacterial or otherwise, is the cause, notwithstanding the absence of the cellular elements, which would justify us in regarding the disease as inflammatory. Certainly long casts may be seen in other conditions than a pure mucous colic, *e. g.* occasionally in cases of carcinoma and of ulcerative colitis. Whether or no we regard it as a pure neurosis, I am sure that the nervous state must be reckoned with in any attempt at curative treatment.

During an attack of mucous colic the treatment is plain. As a rule castor oil is sufficient. Seldom are any injections necessary; if anything is necessary in this way, six or eight ounces of warm olive-oil containing cocaine may be of use. Very rarely is morphia necessary. But the severity of the pain, the frequency of the attacks, and the wearing effect on the patient have, in a few recorded cases, led to a colostomy. The real difficulty lies in the treatment of the patient between the attacks, and the difficulty is increased if the attacks occur only at long intervals. Perseverance in treatment is not a strong feature in such patients. Constipation comes first, and it requires the usual line of treatment by diet, by a long course of abdominal massage, by exercises, and by the determined diminution and ultimate withdrawal of purgatives. I have faith in the use of olive-oil, regulin, and agar-agar. I have never satisfied myself that the stringent coarse cellulose diet of Van Noorden can be taken without great discomfort and flatulence. Above all I feel sure that colon-irrigation of all kinds in these pure cases should be avoided if possible. In many of my cases where the pain has been on the right side, and there had been doubt as to its nature, the appendix has been removed. As far as I know, there has never been any resulting improvement. A large part of the treatment must be directed to the general neurasthenic state of the patient. An open-air life and regular graduated exercise under supervision and under the most healthy and cheerful surroundings afford the best chance of improvement. This is easily provided, if only the relatives will consent to part with the patient for two months.

Of one thing I feel sure, viz. that a rest-cure, with all that it means in the way of isolation and nursing, is not to be recommended. I think it is invariably a step downwards, and it robs the patient of what little self-reliance she has. I sometimes see patients who have had several rest-cures at various hands, and who look upon a further series with complete indifference or merely a sense of boredom.

(*b*) A condition also occurs to which no other name than "mucous colitis" can be given. It is as if a secondary bacterial action was in force. It may alternate with pure mucous colic, or it may supervene and replace it, but it may occur without any such connection. Pain is never so severe. The trouble tends to be continuous rather than paroxysmal, and with remissions it may run on for years. From time to time there is the abundant production of mucus, far in excess of anything seen in other forms of inflammation of the colon. In fact, mucus may be passed without fæcal matter. But over and above this mucus-passing, abundant leucocytes, serous exudation, and consequent frequent loose stools are the main feature. Even blood may be seen, though I have never known a case of this kind end fatally with ulceration of the colon. Except by the occasional production of large quantities of mucus, such cases are at times hardly to be distinguished from a chronic catarrhal colitis. But the nervous phenomena and the queer mental state are as constant and characteristic as in mucous colic. Here, again, the nervous side of the ailment requires the same line of treatment as in mucous colic, but it is much more difficult to administer, as the patient's illness is more continuous, and we often have not the intervals of comparative health in which to work.

In obstinate cases some form of flushing must be employed, either by appendicostomy or by the method employed at Plombières, Chatel-Guyon, Harrogate, and elsewhere. I believe that such cases are better without meat, and with no reason to advance I have faith in olive-oil, if only it can be taken in sufficiently large doses. As in the case of mucous colic, I have often known the appendix to be removed without any obvious result. I have no experience of the use of a vaccine in this condition.

April 25th, 1910.

INTERESTING CASES OF ABDOMINAL SURGERY, INCLUDING SOME RARE FORMS OF INTESTINAL OBSTRUCTION.*

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(*Concluded from p.* 28.)

We come now to the two remaining cases (4 and 5) of the series, in which intestinal obstruction, acute or chronic, was caused by a volvulus; in the first, of the small intestine, in the second, of the large intestine.

It may be useful here to make a few general remarks on volvulus. As to the relative frequency Sir Frederick Treves† states that more than two-thirds of all cases are instances of twist of the sigmoid flexure about its mesenteric axis; so that volvulus of the small intestine must be much less common.

"The term volvulus" (of the large or small intestine), he continues, "includes two distinct methods of producing obstruction.

"In Group I, the bowel is so twisted about its mesenteric axis, or even, in rare cases, upon its own axis, that it becomes occluded; in Group II, two suitable coils of intestine are so intertwined or knotted together as to cause also an obstruction in their lumina."

The simple type of volvulus (I), whether in small or large intestine, is illustrated by the diagrams, skilfully prepared from my rough sketches by Mr. Shiells (Fig. 4 [1, 2, 3]), which show the more usual twist, about the mesenteric axis from left to right, the twist in (2) amounting to half a turn, in (3) to a complete revolution.

Case 4, however, is an instance of the very rare form of volvulus (Group II, *supra*), in which "two suitable coils of small intestine become twisted together, the one acting as an axis about which the other is wound, the suitability being due to the loop forming the axis being fixed by its extremity to some point on the parietes."* In the present case the fixation point was the mouth of the duodeno-jejunal fossa, in which the end of the loop became nipped (Figs. 5 and 6).

More commonly, the volvulus depends on the involved loop being possessed of a long and narrow mesentery.

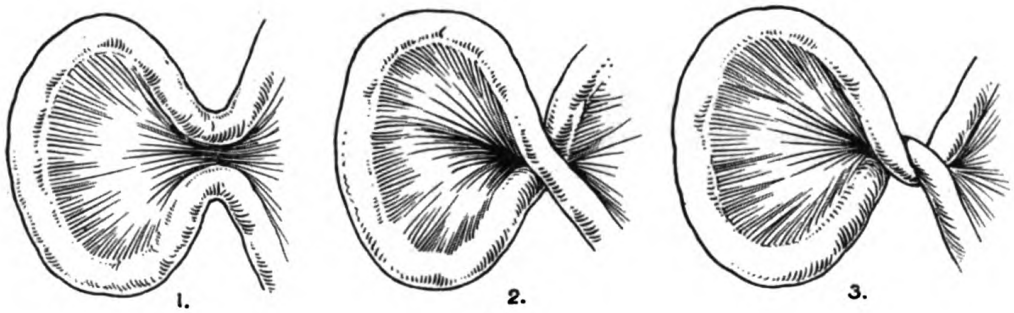

Fig. 4.

Case 4. *Volvulus of the Small Intestine, secondary to a Hernia into the Duodeno-jejunal Fossa; one week's Intestinal Obstruction; Reduction; Cardiac Failure; Death.*

The patient was a young, married, nulliparous, Matabele woman, living at Bulawayo, who was admitted to the hospital under my care at 5 p.m. on February 15th, 1905, for intestinal obstruction, complete for a week, and intense pain in the abdomen referred to a swelling below the left costal margin, moving with respiration, resembling an enlarged spleen, which it subsequently proved to be, the enlargement being probably of purely malarial origin.

There had been no hæmorrhage from the bowel. There had been no vomiting, and none occurred whilst the patient was under observation. Its absence is decidedly unusual in cases of acute or subacute volvulus.

The abdomen was much distended centrally, the

* Read before the Richmond Division of the British Medical Association, on February 23rd, 1910.

† 'Intestinal Obstruction,' p. 126, 1899 edition.

* *Ibid.*

hepatic flexure and transverse colon being apparently also greatly distended and tympanitic on percussion. Both flanks were resonant. The liver dulness was almost obliterated.

Rectal examination was negative, and an enema produced no result. The pulse, 106 per minute, was rapid and feeble. The respiration rate was 42 per minute, the temperature on admission at 5 p.m. being 102·4° F.

Operation was arranged for, and prior to it, to combat the collapse, strychnine and digitalis were injected subcutaneously. Strychnine was required again during the operation, but at its conclusion the pulse was excellent.

Median laparotomy revealed a centrally-placed volvulus of the small intestine, the twist being, as usual, from left to right (Fig. 6).

Recent peritoneal adhesions for a distance of about two inches bound the coils on the right side of the volvulus, at its root or pedicle, to the adjacent posterior abdominal wall. These were readily detached. Examination of the left side of this pedicle then revealed the presence of a long, narrow loop of completely collapsed small intestine, which could be seen running vertically upwards to the left of, and parallel with, the vertebral column, each limb being about six inches in length. The apex of the loop was traced up just into the mouth of a small but well-defined fossa duodenojejunalis.

It became clear that the volvulus had formed as the result of the distended coil, which presented on first opening the abdomen, having become twisted round the axis provided by the long narrow loop herniated into the fossa.

During the manipulations involved in making this examination the herniated loop slipped out of the fossa; by its release and subsequent descent it readily allowed easy reduction of the volvulus.

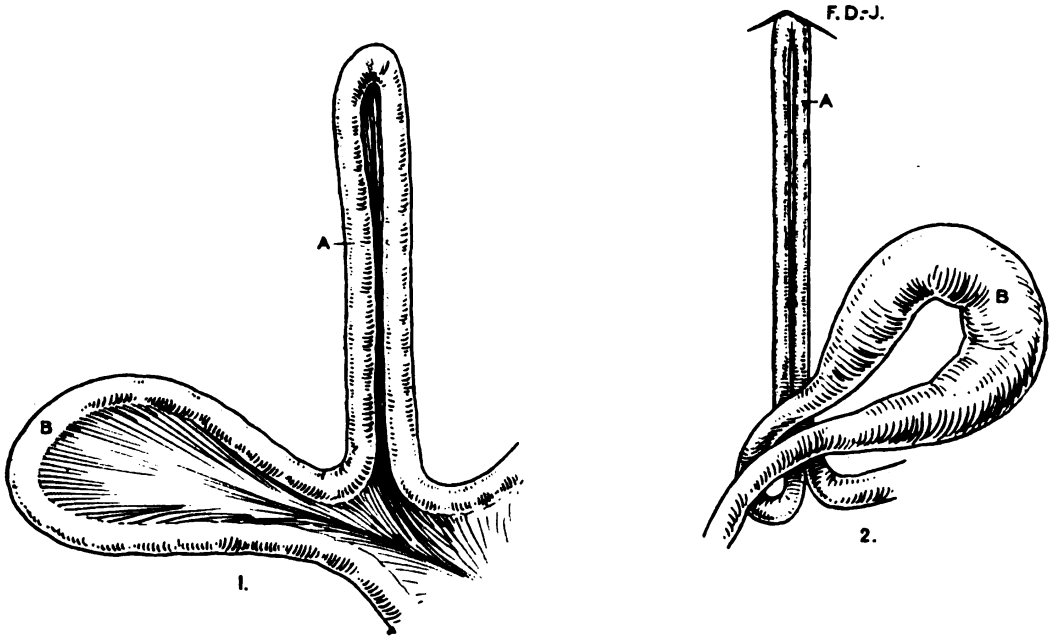

FIG. 5.

The diagrams (Figs. 5 and 6) (also by Mr. Shiells) will serve to illustrate the *modus operandi*.

Subsequent history of the case.—A quarter of an hour after the operation, when the patient was in the ward, the pulse again failed and respiration slowed down, the patient seeming to be moribund.

During the night the pulse and respiration rates per minute were 112 and 32, the temperature falling to 99·4° F. Next morning the bowels were relieved spontaneously. The pulse-rate varied from 112 to 116, the rate of respiration gradually falling from 28 to 24, and then towards the end to 16 per minute. There was no vomiting or other sign of obstruction. Unfortunately, the patient never rallied from the collapse and cardiac failure, in spite of the usual restoratives, transfusion, etc.,

and she died on the day after admission, exactly sixteen hours after the operation, no autopsy being permitted.

From the point of view of relieving the obstruction the operation gave every promise of being a success, the fatal collapse being doubtless due to the week's delay in coming for treatment.

In Liechtenstern's case, quoted by Treves, the axial loop of gut around which the other coil of bowel was twisted was adherent at its apex to the parietes; so that, in effect, it corresponds closely with the present case, which, however, owed its origin to the existence of the fossa duodeno-jejunalis. The formation of a hernia thus provided an axis around which the volvulus occurred.

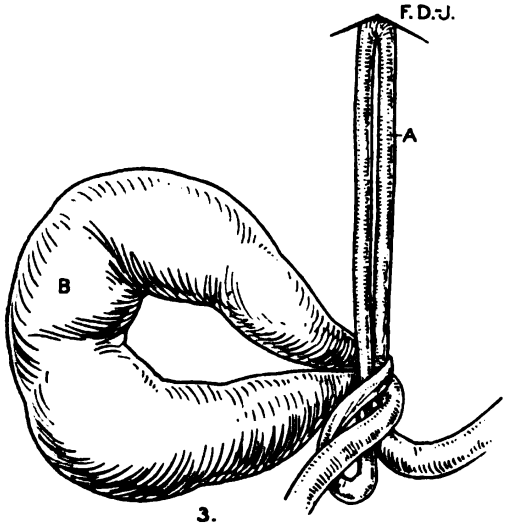

FIG. 6.

The following case illustrates the very complicated arrangement generally resulting from a volvulus of the cæcum and ascending colon. It falls into Treves' third group of twists of this part of the intestine, in which the cæcum is bent upon itself at right angle to its long axis. Treves quotes two examples, and three of the other sub-variety of this group, in which the cæcum is twisted around its own long or vertical axis, so that its relations to the ascending colon are practically undisturbed, and in all five instances, as also in a sixth case referred to, the cæcum presented a congenital freedom of the cæcum and ascending colon.

He adds that these forms of twist must be classed among the least common varieties of intestinal obstruction.

CASE 5. *Volvulus of the Cæcum and Strangulation of the Descending Colon by a Traction Diverticulum, secondary to Chronic Peritonitis, causing Chronic Intestinal Obstruction.*

The patient, a Matabele man, came under my care at the Bulawayo Hospital from a remote district, in 1904.

His white hair indicated a great age, but, as usual among natives, definite information in this respect could not be elicited, all the more so that the patient proved to be a very "raw Kaffir," with whom it was almost impossible to communicate on account of his dialect.

He was admitted for gradually increasing intestinal obstruction, which had become complete during the previous few days.

He appeared to suffer no great pain, his chief trouble being discomfort from distension.

The abdomen was greatly distended, the parietes being much thinned. Peristalsis was obvious, proceeding chiefly from above down in several coils of intestine, two of these being especially evident, extending in a nearly vertical line beneath the umbilicus and in its immediate neighbourhood.

Above these vertically placed coils of bowel was an ovoid prominence, with its long axis horizontal, passing across the epigastric region, which appeared to be transverse colon.

Below this, occupying both flanks, which were greatly distended, were seen bulgings, obviously due to distended bowel, considered to be ascending and descending colon, the vertical coils seen traversing the umbilical region being therefore small intestine.

The percussion note everywhere was resonant, that over the ovoid swelling in the epigastrium negativing the presence of stomach.

Action of the bowels.—As the result of enemata, scybalous masses were evacuated from time to time, but there was no spontaneous relief at any time. The discomfort arising from extreme distension, though relieved temporarily, soon recurred.

Diagnosis.—Abdominal and rectal examination tended to negative the presence of growth in the sigmoid flexure or rectum.

The condition appeared to be one of intestinal obstruction as the result of kinkings by bands from old peritonitis, or, possibly, from the presence of a growth situated in the splenic flexure.

It being certain that continued obstruction, unless relieved surgically, must end fatally, exploratory laparotomy was performed.

Operation.—Median laparotomy through a three-inch incision was performed above the umbilicus.

Two or three vertical coils of congested and greatly distended, but otherwise healthy-looking, small intestine presented in and about the median line. There were no adhesions to the anterior parietes, and none were detected between these various coils.

At the upper end of the incision an opaque, creamy-white, greatly distended piece of bowel presented itself, with its long axis nearly horizontal occupying the epigastric region, corresponding with the ovoid prominence which had been visible there externally.

In spite of the previous assumption, the appearance now strongly suggested stomach rather than transverse colon, especially as the sacculations usually seen in the colon had been almost completely obliterated by distension, and also as the result of long-standing obstruction, the walls of the viscus had become greatly hypertrophied, and lost their characteristic tint. Beneath the left edge of the incision the enormously distended descending colon was seen to be bulging out of the left flank, running vertically down so as to occupy its usual position. The walls were of opaque, creamy-white colour and hypertrophied; the appendices epiploicæ were, however, very obvious. In the apparent absence of evidence of old general peritonitis the distended state of the descending colon was attributed to obstruction below in the region of the left iliac fossa, since no growth could be detected *per rectum*.

The lower half of the abdominal cavity was explored through the original incision, and a firm, stout band, about as thick as the little finger, and about one inch in length, was felt passing horizontally across and constricting the lower part of the descending colon, to become attached by a broad base to the abdominal parietes at a point about one inch above and internal to the left anterior superior spine. The median incision was, therefore, closed, and a fresh vertical incision, about $3\frac{1}{2}$ in. long, was made over the centre of the left rectus muscle. The incision extended to about 1 in. below the left anterior superior iliac spine, and the stout band felt previously was now found immediately to the left of the new incision, located as just described.

When brought into view this band exactly resembled the terminal inch or so of the appendix, somewhat enlarged and bulbous at its tip, so as to be pear-shaped at the outer extremity, which was very firmly adherent to, or rather incorporated with, the parietal peritoneum, at a spot above and internal to the left anterior superior iliac spine.

The band appeared to consist either of the appendix drawn horizontally across from the right side of the abdomen, or an adherent diverticulum.

After division of the band between two ligatures it was found to contain a lumen lined with mucous membrane, in every respect, in size and general appearance, resembling that found on dividing the appendix, or a very narrow diverticulum.

On following it up it was seen to pass transversely from left to right between coils of the small intestine. An attempt to trace the diverticulum, after its division to its origin had to be abandoned almost at once, however, as in attempting, with the utmost care and gentleness, to separate it from adhesions to surrounding coils of intestine, a thinned portion of bowel somewhere gave way, with resulting exit of a little gas. The perforated spot was brought into view and was readily closed, a piece of congested great omentum lying near being sutured over it. This piece of omentum at the autopsy was found to be extending from the greatly distended transverse colon so as to be attached to the small intestine.

The exact nature of the diverticulum (Meckel's or other form) was therefore not determined at the operation.

The immediate effect of dividing the constriction thus formed was to cause the speedy collapse of the distended descending colon.

As nothing else was detected by digital examination through the lower incision, in the apparent absence of general peritonitis, and the general healthy though distended appearance of the small intestine, it was hoped that the distension, evidently chronic, was entirely due to the constricting diverticulum, now divided. The abdominal wound was therefore closed.

Subsequent course.—Death occurred a few days later without any relief of the obstruction, and with steadily increasing distension.

Autopsy.—The peritoneal cavity was freely laid open by a median incision from sternum to pubes, and later by a cross-cut connecting the iliac spines, owing to the extreme difficulty experienced, even now, in determining the condition of things present. To have made a completely correct diagnosis at the time of operating, therefore, was almost impossible.

Inspection of the diagrams (Figs. 7 and 8) prepared from sketches I made at the time, after prolonged post-mortem examination, and thorough exposure of the small intestine by dividing adherent great omentum, will, however, greatly simplify the description. It must here be recalled that with the division of the diverticulum strangulating the descending colon at the operation this portion of the bowel at once collapsed, and at the autopsy remained completely concealed by small intestine.

Drawing an oblique line across the abdomen from the right anterior superior iliac spine to the opposite left costal margin, about the level of the ninth rib, the parts presented below, and to the left of this line, consisted entirely of distended small intestine. One greatly distended **S**-shaped coil passed from the right iliac fossa, almost vertically upwards from the mid-Poupart line to the umbilicus, above which it crossed over to the left costal margin to become lost beneath the left flank. From below the arch of bowel thus formed two vertical loops of small intestine proceeded (from the back of the abdominal cavity) forwards and downwards, to disappear beneath a series of five or six loops,

horizontally placed, of small bowel, which, as stated, completely concealed the splenic flexure, the descending portion, and the sigmoid flexure of the colon.

Occupying the epigastric region and extending to the right, well into the hypochondriac region, was a piece of bowel, so distended and so altered in contour and consistence as even now, at first sight, to be mistaken for a distended stomach. It proved, however, to be the left end of the transverse colon, the stomach itself being concealed and lying collapsed deep down behind it. Adhesions, consisting of bands of congested great omentum, passed from the left end of the transverse colon to the horizontal coils of small intestine shown in the diagrams, and as they were continuous with the congested adherent omentum covering the site of the diverticulum divided at the operation, they were probably of subsequent origin. They required to be divided before the view presented in the diagrams could be obtained, showing the horizontal arch of the S-shaped loop of small intestine already described.

Extending well up to the right costal margin, and occupying the entire right lumbar region and its junction with the adjacent umbilical region, the cæcum was seen, enormously distended, lying with its longer (usually almost vertical) axis horizontally placed, being, in fact, in a condition of volvulus, and acutely kinked upward on itself, so as to conceal practically the whole of the ascending colon, the kinking resulting from firm old adhesions of the congested and thickened great omentum.

These adhesions, so high up and close beneath the right costal margin, were not detected during the operation, and even at the autopsy the appearances presented were so unusual as almost to baffle explanation.

The cæcum, thus kinked up, lay with its anterior longitudinal muscular band (the anterior tænia coli) running horizontally outwards to the right, and by following this onward and thoroughly retracting the abdominal wall flaps, the caput cæcum coli and the appendix were discovered, the appendix being nearly six inches long, full of mucus, but otherwise healthy-looking, running upwards deep in the flank, on the outer side of the ascending colon, and ending at about the level of the hepatic flexure.

Occupying the right iliac fossa, between it and the cæcum, was the greatly distended terminal portion of the ileum, passing outwards, upwards, and backwards, to connect up with the posterior aspect of the displaced cæcum.

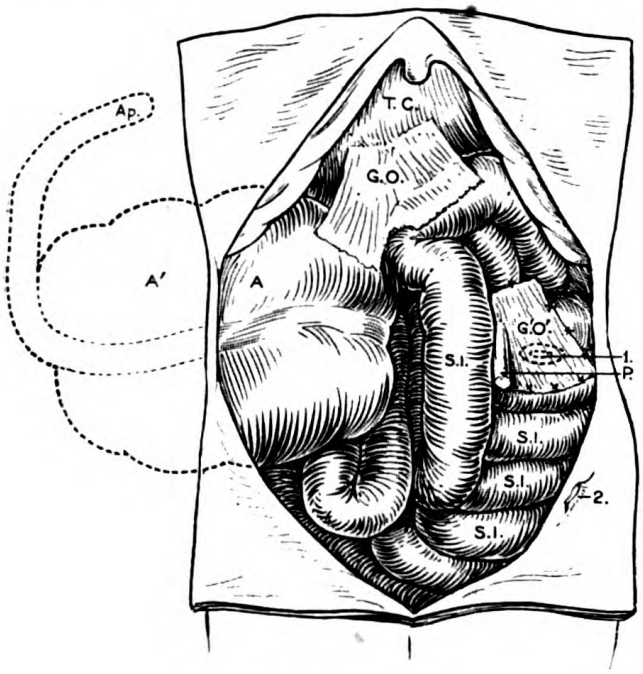

Fig. 7.

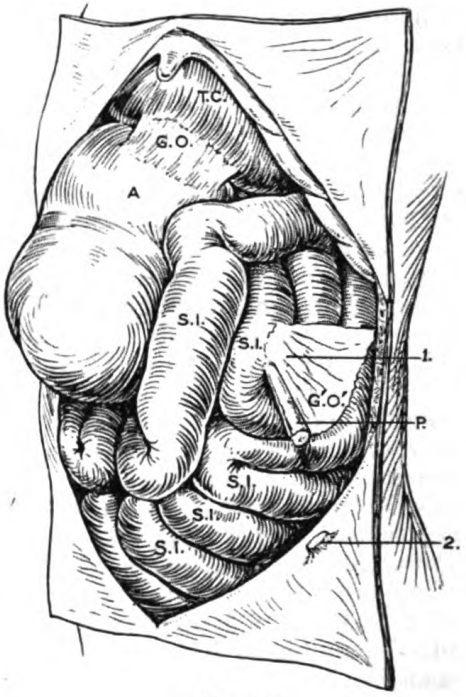

Fig. 8.

The volvulus of the cæcum thus occupied the right lumbar and lower part of the right hypochondriac regions, the remainder of the right hypochondrium and most of the epigastrium being occupied by the transverse colon, whilst the usual situation for the cæcum, the right iliac fossa, was filled up with greatly distended ileum.

The ileo-cæcal valve was felt to be blocked up by irregular hard masses of bony consistence, examination of which subsequently indicated that the patient must have been fond of poultry, and bolted many a claw, numerous metatarsals, etc., and larger fragments of bone, being easily seen in the specimen bottle I now show you (Fig. 9).

They doubtless played an all-important *rôle* in the ætiology of the disease.

Examining now the site of the operation, it was found to be attached to the anterior abdominal wall by a thin band of peritoneal adhesion, evidently post-operative. A triangular patch of congested omentum covered the region where the proximal extremity of the detached diverticulum lay. The

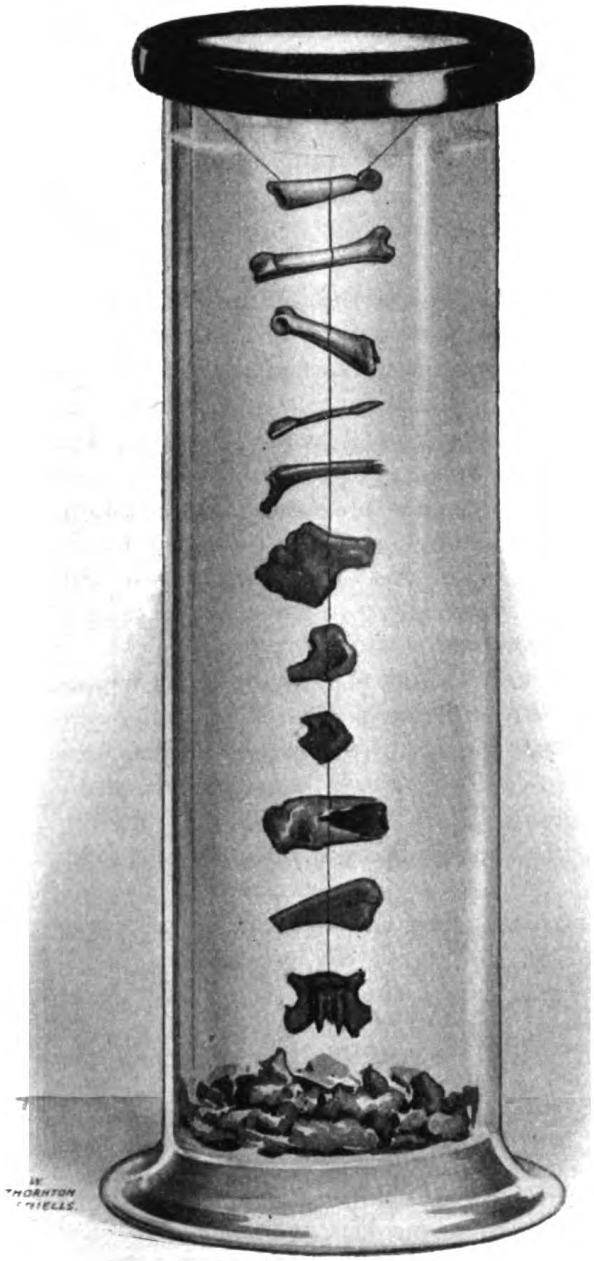

Fig. 9.

puncture seemed to have been successfully sutured. The original diverticulum could not be satisfactorily traced to its origin, and though it was possibly a short Meckel's diverticulum, it much more probably originated in a small pocket resulting from the small intestine becoming adherent at one edge to the fossa, and by subsequent peristaltic movements becoming a so-called "traction-diverticulum," which, by adhesion to the left iliac fossa, blocked the descending colon. A roughened area in the left iliac fossa, lozenge- or diamond-shaped, indicated the spot from which the diverticulum was detached at the operation (see Figs. 7 and 8).

The presence of the numerous fragments of bones impacted above the ileo-cæcal valve would seem to indicate the exciting cause to have been the undigested and sharp fragments of bones, which, setting up a chronic peritonitis and inflammation of the adjacent great omentum, resulted in the adhesions and strangulating traction-diverticulum. It seems certain, however, that here, as in the cases quoted by Treves, the predisposing cause of the volvulus of the cæcum was an abnormally long meso-cæcum of congenital origin.

In conclusion, it is interesting to note that the Conservator of the Royal College of Surgeons' Museum, Professor Arthur Keith, F.R.C.S., who has recently* been lecturing on the anatomy of the negro and negroid races, informs me that amongst negroes the pelvis, both male and female, is smaller than in white people; and in consequence of this, probably, the cæcum lies considerably higher up in the abdomen than with us. He does not believe, however, that, generally speaking, there is a greater tendency than with us to an abnormally long meso-cæcum, causing greater freedom of the cæcum and ascending colon.

April 25th, 1910.

A CASE OF MYOMECTOMY DURING PREGNANCY.

By FRANK E. TAYLOR, M.A., M.D., M.Sc., F.R.C.S., M.R.C.P.,

Gynæcologist to the Hampstead and North-West London Hospital; Obstetric Registrar and Tutor to the Middlesex Hospital.

The remarkable tolerance which the pregnant uterus displays to operative manipulations fully bears out the aphorism that the healthy ovum adheres to the healthy uterine wall with marvellous tenacity, and is well illustrated by the history of the following case, in which three sub-peritoneal fibroid tumours were removed from the uterus on account of pain about the middle of pregnancy without interfering with the course of the pregnancy.

In April, 1909, I was asked by Dr. Stubbs, of Brixton Hill, to see a married woman, æt. 36 years, complaining of a painful and tender abdominal swelling associated with amenorrhœa.

* February 14th–21st, 1910.

The patient had been married ten years and had never been pregnant. Menstruation had always been quite regular and painless until December, 1908, since when there had been complete amenorrhœa.

In January, 1909, a lump was felt in the abdomen, which had been gradually getting larger since. There was some sickness during December and January, but this usually occurred towards evening and not in the mornings. The breasts, too, at first enlarged, but the patient thinks they had lately somewhat diminished in size. A well-marked areola was present, with Montgomery's follicles, and a little serous fluid could be expressed.

The mass was free from pain until a few days before I saw the patient, but for the last few days there had been considerable pain in it.

Inspection showed the abdomen to be generally enlarged, especially below the umbilicus, with definite bulging in the left flank. On palpation two definite swellings could be made out, one slightly to the left of the middle line, reaching halfway up to the umbilicus; it was softish in consistence, giving the impression of the pregnant uterus, and was free from tenderness. The other swelling lay to the right of this and bulged the right flank; it was hard and very tender and was freely movable, and appeared to be attached by a pedicle to the uterus. The pain was definitely located to the site of this swelling. A uterine souffle could be plainly heard on auscultation, but fœtal heart-sounds could not be detected.

On vaginal examination the vulva and vagina showed distinct violet discoloration. Bimanually the cervix was softened, and was continuous with the softish tumour above described on the left of the abdomen, which was free from tenderness. On the right the lower pole of the hard abdominal tumour could only be felt; it was freely movable and very tender. The pulse and temperature were normal. I came to the conclusion that the softish swelling reaching half-way to the umbilicus was the pregnant uterus a little over four months, and that the hard, movable, tender swelling to the right of the uterus was a pedunculated subserous fibroid undergoing red degeneration, which accounted for the pain and tenderness.

On account of the patient's age, the fact that she was pregnant for the first time after ten years of married life and that she herself was very anxious to have a living child, I recommended no interference, and that the patient should go to term and be delivered naturally, seeing that the fibroid was well out of the pelvis, and would not cause any obstruction to the passage of the child.

The pain and tenderness, however, increased in severity to such an extent that the patient demanded that something should be done even if it entailed loss of the child.

She was accordingly admitted to the Hampstead General Hospital on May 12th, 1909. The condition was still the same as that described except that the uterus now reached almost to the level of the umbilicus.

On May 15th I opened the abdomen by a median sub-umbilical incision, and the enlarged uterus, soft and bluish, presented. Attached to the right side of the uterus was a pedunculated fibroid tumour about the size of a fœtal head. Omentum and intestines were found attached to the whole convex surface of the tumour by recent soft adhesions, These were readily separated with the finger. The pedicle, which was of the thickness of two fingers, was ligatured by transfixion and the tumour was removed.

Two sessile subserous fibroids, the size of a plum and a small tangerine orange respectively, were found in the fundus of the uterus. These were enucleated and the spaces were obliterated by catgut sutures. The abdominal wall was sutured in three layers, the skin being united by Michel's clip sutures.

Recovery was uninterrupted, the sutures being removed on the eighth day, when fœtal heart-sounds were heard and the patient felt fœtal movements.

Dr. Torrens, the pathologist, reported on the specimen as follows: "The tumour is a very necrotic fibro-myoma, suggesting that the blood supply has been interfered with." There had, however, been no torsion of the pedicle, nor was the condition a true red degeneration, the tumour being softish, grey in colour, and containing a few small cystic spaces containing clear fluid.

The pregnancy went to term, and Dr. Stubbs delivered the patient of a healthy girl weighing 9½ lb. after an extremely easy labour, lasting only four and a half hours, the abdominal wound remaining sound and firm.

It is interesting to note that an elder unmarried sister who lived with the patient had been known to suffer from fibroids for several years.

April 25th, 1910.

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CONTENTS.

PAGE

*A Clinical Lecture on Fibrositis of the Abdominal Muscles. By GEORGE R. MURRAY, M.A., M.D., D.C.L., F.R.C.P. 49

*Medicine on the Continent. By HARRY CAMPBELL, M.D., F.R.C.P. 53

*On Two Common Errors in the Administration of Anæsthetics. By J. D. MORTIMER, M.B., F.R.C.S. 63

* *Specially reported for the Clinical Journal. Revised by the Author.*

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A CLINICAL LECTURE

ON

FIBROSITIS OF THE ABDOMINAL MUSCLES.

By GEORGE R. MURRAY, M.A., M.D., D.C.L., F.R.C.P.,

Professor of Systematic Medicine, University of Manchester; Physician to the Manchester Royal Infirmary.

WHEN we consider how large a proportion of the weight of the whole body is made up by the muscles it is rather surprising that so little attention is paid to their common diseases. The rare diseases of the muscles, on the other hand, have attracted a considerable amount of attention as they are of great pathological interest. It is only necessary to mention such uncommon muscular maladies as myositis ossificans, the various forms of idiopathic muscular atrophy and hypertrophy, and Thomsen's disease, to remind you how rarely these interesting conditions are seen in practice. It is, however, not to these that I wish to direct your attention to-day, but to the much more common cases of acute, subacute, and chronic inflammation of the muscles, which are commonly called muscular rheumatism, interstitial myositis or fibrositis, a very convenient term for which we are indebted to Sir William Gowers.*

These, in one form or another, are so common that we all come across them from time to time, and I fear that few of you will pass through life without experiencing the symptoms in one muscle or another. Well-recognised types of the malady, such as lumbago, are of course familiar to all, and their recognition is easy, but many other diseases may be simulated by fibrositis occurring in unusual situations, and it is for this reason that I thought the subject was worthy of your consideration.

* 'Brit. Med. Journ.,' January 16th, 1904, p. 117.

The muscles form a very large proportion of the total weight of the body. It was estimated by Dr. G. v. Liebig * that in a man weighing 150 lb. the muscles weighed 63 lb., the skeleton 27 lb., and the viscera (including skin, fat, blood, etc). 60 lb. Thus the muscles actually weigh more than all the other organs in the body put together, and yet in one of the leading text-books of the day only four out of 1114 pages are devoted to the consideration of their diseases. There are, of course, good reasons for this. The muscles are not vital organs, and only a few of the rare forms of muscular disease are dangerous to life, nevertheless a great deal of suffering is caused by muscular diseases, and they are well worth more attention than they usually receive, as those of you who have had the misfortune to suffer from lumbago will readily realise. Our knowledge of the pathology of fibrositis is unfortunately very scanty, but it is well worthy of attention, as further knowledge might assist us in the prevention and treatment of these common maladies.

Every muscle consists of a mass of muscular fibres, which are surrounded and bound together by fibrous connective tissue, the perimysium, and sheathed by the epimysium. Many muscles are connected by means of tendons to bones. It is to the inflammatory conditions which occur in the fibrous tissue of the muscular and tendinous portions of the muscles that the name "fibrositis" has been given, and to which I wish more especially to draw your attention.

The muscles are provided with sensory nerves, which are connected with the muscle-spindles which lie in the fibrous tissue between the muscular fibres. Under ordinary circumstances the sensory impulses which pass up to the brain from these curious structures are unperceived by us. They nevertheless register the amount of contraction which takes place in the muscle, and when this is excessive, as in "cramp," the muscle spindles are strongly compressed and give rise to acutely painful sensations. In fibrositis these structures become much more sensitive than in health, so that even an ordinary amount of contraction or tension of the muscle affects them in such a manner as to give rise to very painful stimuli, which, though different in degree and duration, are similar in character to those which are caused by "cramp." There are some 311 voluntary muscles in the body differing greatly in size and shape, and the relative amount of muscular fibre and connective tissue in each. Thus it is easy to understand why such a variety of symptoms may occur according to the special muscles which are involved.

Causation.—The causes of muscular rheumatism are still obscure. The term "rheumatism" is misleading, because there is no evidence to show that it is infective in origin. Assuming that the *Micrococcus rheumaticus* is the cause of acute rheumatism, we have no evidence that it plays any part in fibrositis. Men are much more liable to be attacked than women as they are more exposed to changes of temperature and to muscular strain. There can be no doubt that exposure to cold and wet plays an important part in inducing an acute attack, as, for example, of the "muscular" form of lumbago. Gouty subjects are especially prone to suffer, and it is probable that certain articles of food and drink predispose to the malady; thus claret is particularly liable to lead to the development of recurring attacks of lumbago. In some cases one or two glasses of claret taken each evening with dinner have been quite sufficient to induce and maintain a liability to lumbago, which disappeared as soon as this wine was no longer taken.

One form of fibrositis results from acute or chronic strain. An acute strain may be caused by a sudden contraction of a muscle, as in attempting to recover the balance after a slip or by sudden tension of a muscle by over-extension of a joint. A chronic strain may be due to some habitual posture which is necessitated by the occupation. Thus the back-ache which so many coal-miners suffer from is probably due to a chronic form of tendinous lumbago. The effects of prolonged or repeated strain appear to produce more persistent symptoms in tendinous than in muscular structures, as the former not infrequently prove very intractable to treatment. The whole question has assumed a greater importance since the Workmen's Compensation Act came into operation. In some of these chronic cases it is by no means easy to determine the causation of the symptoms complained of, and to determine their true relationship to some alleged accident or strain which has occurred in the course of the patient's occupation.

* Quoted in 'Quain's Anatomy,' vol. ii, pt. 2, p. 201.

As the symptoms may be purely subjective the difficulty is often great, and these cases are not infrequently brought into court for trial.

Symptoms.—The chief and most characteristic symptom of fibrositis is pain. The situation of the pain depends upon the particular muscle or tendon which is affected, so that pain in almost any part of the body may be due to this cause. The character of the pain varies. It is usually a constant, dull, aching pain, which is only relieved by complete relaxation and rest of the affected muscle. It is brought on or aggravated by continuous or irregular contractions or by putting the muscle on the stretch, and when induced in this manner it may be extremely severe for a time until relief is obtained by rest and relaxation. Thus, for example, in some forms of lumbago the pain is induced or increased by the continuous contraction of the lumbar muscles in sitting or standing in the upright position, or by the varying contractions which occur in walking or in sitting in a railway carriage on a long journey. An acute pain in the lumbar region is often experienced on rising up after sitting at a table writing, that is to say, on contracting the lumbar muscles after they have been stretched by the flexed position of the trunk assumed in writing. In the more chronic forms the pain may not come on at once, but only when the muscle has been in ordinary or extraordinary action for some length of time.

There is no superficial or cutaneous tenderness, but deep pressure over the muscle, especially when it is strongly contracted, elicits tenderness at one or more points. This is a valuable test, especially in the case of abdominal muscles. If, for example, the pain complained of is in the region of either of the recti abdominis muscles, it may be found, when the patient is examined lying flat on the back on a couch, that there is no marked tenderness on pressure over any part of these muscles. If the patient is asked to raise the trunk until its long axis forms an angle of forty-five degrees with the surface of the couch, so that the recti muscles are strongly contracted, it will be found on pressing the tip of the finger firmly into the contracted muscle, at the point where the pain is usually felt, that it is acutely tender. These localised areas of tenderness in the muscle during strong contraction are of much assistance in distinguishing fibrositis of the abdominal muscles from intra-abdominal conditions. In intra-abdominal disease the tenderness is complained of when pressure is made over a relaxed muscle, or there may be a reflex contraction of the muscle which prevents deep pressure being made, whereas in fibrositis there may be but little tenderness when the muscles are flaccid, but acute tenderness on pressing firmly on the muscle when it is in active contraction.

On careful palpation of the affected muscle a localised swelling can in many cases be distinctly felt. This is the more readily to be made out if the edge of the muscle can be grasped, as, for example, when it occurs along the upper border of the trapezius. In some acute forms of lumbago a distinct swelling can be felt in the lumbar muscles. When the tendinous portion of the muscle is chiefly affected no swelling may be detected. In many of these cases the pain is aggravated by the feeling of apprehension of the patient, who fears that it is an indication of some serious internal malady. When, after a careful examination, he can be assured that his pain is entirely due to fibrositis of an abdominal muscle, the mental relief is great, and I have been impressed by the cheerfulness with which pain is borne as soon as it is realised that it is due to a simple cause which will subside.

Fibrositis of the thoracic or abdominal muscles is not an uncommon variety of the malady, and is apt to simulate intra-thoracic or intra-abdominal disease. In order to understand the symptoms it produces I must shortly remind you of the anatomy of the abdominal muscles. The muscular wall of the abdomen on each side is composed of three layers, the fibres of the two outer layers being oblique and of the innermost being transverse in direction. The external oblique arises from the outer surface of the eight lowest ribs, and is inserted partly into the anterior half of the outer margin of the crest of the ilium and partly into the aponeurosis. The internal oblique arises from the outer half of the deep surface of Poupart's ligament, from the crest of the ilium and from the lumbar fascia. Its fibres, spreading over the lateral aspect of the abdomen, are inserted into the three lowest ribs. the aponeurosis on the front of the abdomen, and by means of the conjoined tendon into the symphysis pubis. The transversalis arises from the inner side of the six lower rib-cartilages, from the transverse processes of the lumbar vertebræ, from the inner edge of the anterior two thirds of

the iliac crest and the outer third of Poupart's ligament, and is inserted into an aponeurosis in front and the conjoined tendon below. The most important muscle is perhaps the rectus abdominis which arises by a tendon from the pubes below to be inserted into the cartilages of the fifth, sixth, and seventh ribs above. Not only has this muscle a tendinous sheath and origin but it has several tendinous intersections. The three most important of these are placed—one opposite the umbilicus, one on a level with the lower end of the ensiform cartilage, and the third midway between the two. This muscle, like the lumbar muscles, is apt to be involved in fibrositis on account of its anatomical connection and liability to strain.

Though not dangerous to life in itself an acute fibrositis may in some enfeebled states of health contribute to a fatal issue in consequence of the very severe pain it may produce. Thus a lady, æt. 66 years, had a mild attack of influenza. Three weeks after this, while engaged in household duties one day, she did a good deal of stooping, in consequence of which she complained of aching in the back on that and the following days. Four days later she was seized with an acute pain in the back just above the upper border of the sacrum on the right side. The pain radiated round to the front of the lower part of the abdomen and into the right groin, and so resembled the pain of renal colic. There was, however, neither fever, shivering, nor sweating. Some nausea was complained of, but there was no vomiting. On examining her with her own medical attendant we found there was a very tender spot about the size of a florin just above the upper border of the sacrum on the right, and when the lumbar muscles were put on the stretch pressure over this point caused an acute pain, and was evidently due to a localised area of fibrositis. The patient was greatly exhausted by the pain, the pulse was 120, and there were signs of cardiac muscle failure. She rallied for a few days, but then died from heart failure. In this case, in which there was a chronic myocardial degeneration, the final cardiac failure was apparently much hastened by the acute pain of the fibrositis of the lower part of the quadratus lumborum.

Muscular pain in the right lower abdomen may give rise to the idea that it is due to appendicitis. Thus a gentleman, whose sister had recently suffered from an acute attack of appendicitis, had been punting on the Thames. He came for an opinion as to the cause of a pain which he had in the right iliac region and which he thought indicated appendicitis. In this case there was no tenderness on moderate or deep pressure in the region of the appendix when theabdominal muscles were relaxed, but as soon as the muscles were made to contract there was marked tenderness on pressure being made on the muscle in this situation. There were no other symptoms, and the pain was due to a localised fibrositis in the right external or internal oblique muscle due to a strain. In another case, a man, æt. 25 years, complained of a pain in the right side of the lower part of the abdomen which had troubled him for the last two years. There was tenderness on pressure along the outer border of the right rectus muscle from the level of the umbilicus to a point three inches below this. When half sitting up so that the recti muscles were thrown into strong contraction, this part of the outer border of the right rectus was acutely tender on pressure, the long-continued pain being due to a chronic fibrositis of the outer part of the lower segment of the right rectus in which the sheath of the muscle was probably also involved.

When fibrositis gives rise to pain in the right hypochondriac region it may be mistaken for biliary colic. Thus a man, æt. 60 years, consulted me on account of pain in the right hypochondriac region which had troubled him for four years, and had been attributed to the presence of a gall-stone. There had, however, been neither shivering, sweating, vomiting, nor jaundice associated with the pain. There was some tenderness on pressure on the outer border of the upper part of the right rectus muscle. When this muscle was firmly contracted the tenderness on firm pressure at the spot was acute. The pain was induced by walking, and was sometimes associated with pain in the muscles of the back. In this case there was no real evidence of gall-stones, and the symptoms were clearly due to fibrositis of the right rectus muscle.

Pleurisy may be simulated by fibrositis, either of intercostal or abdominal muscles. In one such case a medical man was seized with acute pain over the lower part of the front of the left side of the chest, which was much increased on taking a deep breath. On examination no sign of pleurisy was found, but it was noted that the pain was situated just over the insertion of the left rectus

abdominis into the rib-cartilages of the fifth, sixth, and seventh ribs, and was greatly intensified when this muscle was made to contract strongly by rising to the sitting position or by putting it on the stretch by elevating the ribs in a deep inspiration. The symptoms were therefore due to a fibrositis of the upper portion of the left rectus muscle and its tendon. In a similar manner fibrositis of one or more intercostal muscles may also simulate pleurisy.

Treatment.—In acute cases of fibrositis complete rest in bed is required, so that the affected muscle may be kept at rest in a relaxed position and the surface over it kept at an even temperature. At the onset of an attack a hot vapour bath, so as to induce free perspiration, is very valuable, and may cut short an attack in the early stages. The many forms of cabinet baths now on the market render the application of this treatment at home quite simple. If, however, such a bath is not available, a simpler plan, which was described to me by a hospital patient who suffered from chronic Bright's disease, may be adopted instead. This man fixed one end of a piece of hose-pipe to the spout of a kettle which was placed on the fire and the other to the rose of a watering can, which was tied underneath the seat of a wooden chair. He then sat on the chair with a blanket pinned round his neck so that it covered him and the chair like a tent. As the kettle boiled he obtained quite a good and inexpensive vapour bath.

During the acute stage five grains of aspirin or ten grains of sodium salicylate given every four or six hours, and the local application of a liniment composed of equal parts of camphor, chloral hydrate and menthol, as recommended by Dr. Luff, will prove most useful. The efficiency of the liniment is increased if (after it has been applied) some dry hot flannel is laid over the painful area and the heat maintained by means of a hot-water bag. If the attack occurs in a gouty subject, half a pint of hot water with a slice of lemon should be taken night and morning, and an alkaline mixture with colchicum wine should be prescribed. A mixture containing sodium bicarbonate with tincture of belladonna has been found useful in some cases. In the more chronic cases iodide of potassium in small doses of from three to five grains may be given with or without sodium salicylate. If the iodide is not tolerated well guaiacum resin in ten-grain doses may be given three times a day in milk, In some cases ammonium chloride in twenty-grain doses thrice daily, in others tincture of cimicifuga, which should be prescribed with a few minims of liquor potassæ to prevent precipitation, have proved to be of service. In these cases gentle massage or the application of iodine and belladonna ointment until the skin is well reddened is of much value. Massage is particularly useful in cases of muscular fibrositis in which areas of induration can be felt in the substance of the muscle. In obstinate cases a course of treatment by douche-massage or radiant heat at Harrogate or Buxton often serves to remove the last traces of an attack, which otherwise are apt to linger on indefinitely.

May 2nd, 1910.

MEDICINE ON THE CONTINENT.*

By HARRY CAMPBELL, M.D., F.R.C.P.

HAVING recently been so fortunate as to visit a number of the leading European hospitals, under especially favourable auspices, I believe that a record of some of my experiences and impressions may not be without interest to the medical profession in this country. The journey was undertaken in conjunction with my lamented colleague, Dr. Savill, who arranged our programme with such forethought and care that we were received at the appointed times at no less than twenty-three institutions, and were able to interview some sixty medical men, all of whom showed us the utmost courtesy and kindness.

It was gratifying to find that English medicine is held in high repute on the continent. The names of our leading men are all well known there. The German physicians were especially loud in praise of the English school of physiology, while in the experimental physiology of the nervous system there seemed to be a general consensus of opinion that the English lead the way, the names of Langley and Sherrington being singled out for special distinction in the domain of neurology.

* Dr. Campbell was one of the Commission appointed by the management of the West-End Hospital to inspect and report on hospitals for the treatment of nervous disorders.

As a preliminary remark, I may state that there are no free beds in continental hospitals. Although the buildings are erected by the State, and are largely maintained by public funds, every patient has to pay, directly or indirectly, according to his means. If his poverty is such that he is unable to find even the small sum necessary for his admittance, it can be claimed from the local authority under whose jurisdiction he lives.

It will be convenient to let my remarks follow the line of our route, and I thus begin with Paris.

In spite of the great part which the city of Paris has played in the history of medicine, she is much behindhand in the matter of hospital construction. Soon, however, La Pitié will be completed, and "là ville lumière" will then have a hospital worthy of her traditions. A visit to the Salpètrière, practically the same to-day as it was in Charcot's time, emphasises the fact that it is the man and not the building which makes a medical school famous, or, as Professor Babinski aptly put it, "the bird, and not the cage, is the all-important consideration."

The wards date back to the middle of the eighteenth century, while the school buildings, laboratories, etc., are of the humblest description, in some cases little more than temporary sheds. Charcot's library, which contains a considerable number of English volumes, has been preserved with reverent care much as he left it.

Dr. Raymond, the present professor of neurology at the Salpètrière, is a brilliant teacher, and enthusiastically devoted to his work. Dr. Vincent, one of his assistants, has of late been engaged in some interesting observations on the changes which the third nerve undergoes in tabes dorsalis. From three patients he has examined as many as two thousand sections of this nerve, and in all of them he has obtained evidence of a progressive sclerosis. The sections I examined exhibited this change in a marked degree. Like Prof. Raymond, this observer attributes the sclerosis to a localised meningitis starting at the base of the brain.

In sharp contrast with the Salpètrière, perhaps all round the most up-to-date hospital in Paris is the Ophthalmic Hospital, known as the Fondation Roschilde. So palatial is this hospital that its lavishness almost provokes criticism. One feels that the designer has been too prodigal of his space and resources: a much smaller building would certainly have sufficed for its eighty beds. It is evident that his great aim has been to secure complete asepsis, but the idea would appear to have amounted to a veritable obsession, as it has with more than one other architect of recently constructed hospitals on the continent. All the walls are lined with glistening tiles of the purest white, so as readily to show the smallest speck of dirt: the ceilings are white, the floors are white, everywhere is the same tiring monotony of surface, unrelieved by the slightest note of colour. Now cream colour is equally clean-looking, and a soft pale green is far less fatiguing to the eye, and one asks if absolutely satisfactory aseptic conditions demand such complete sacrifice of the æsthetic sense. In conformity with this idea in the artist's mind, the minutest—I had almost said the most extravagant—precautions have been taken against the collection of dirt by a rounding of all corners, and the exclusion of everything in the shape of curtains, mats, cushions, and even flowers. In this hospital we find, indeed, the very apotheosis of cleanliness, but at how great a sacrifice of beauty, and, I would add, of homeliness and comfort as well! The operating room is of enormous dimensions, and is lit by a gigantic window which takes up the whole of one side. What good purpose is served by having so large a room for operating upon so small an organ as the eye I am unable to say.

Another institution we visited in Paris was the "Sanatorium for the Treatment of Nervous Disorders and Morphinomania," presided over by Dr. Sollier. This institution is fitted up with all the most modern appliances for the treatment of diseases of the nervous system—electrical, mechanical, hydropathic, and gymnastic. The chief affections treated are neurasthenia and morphinomania. There are three methods of cutting down the morphia dose in dealing with the latter affection—the sudden, the quick, and the gradual.

The method adopted by Dr. Francis Hare at the Norwood Sanatorium, where I have had the opportunity of following the treatment in a number of cases, is a combination of the first and third. He at once reduces the quantity of morphia to one half, and then proceeds with its further diminution. Dr. Sollier's plan is by rapid reduction; by the end of the first week the patient's supply is entirely cut off. He writes: "It is now acknowledged by

all physicians competent in regard to morphinomania that the best treatment is that by rapid curtailment."* In his opinion the great object to be aimed at is the stimulation of the excretory organs by purgatives, diuretics, sudorifics, etc. He strongly disapproves of giving "sedatives and substitutes," such as bromides, chloral, and, above all, sulphonal, which he contends may do great harm by checking excretion.

A visit to the medical institutions of Paris would have been very incomplete had it failed to include the Pasteur Institute. Here we were received by Metchnikoff, the successor of the immortal Pasteur. He received us in his laboratory, the self-same one, as he proudly told us, that he has worked in for the past twenty years, and where he had just been giving a sitting to an artist. As is well known, Metchnikoff is, with Ehrlich, of Frankfurt, one of the recipients of the recently conferred Nobel prize.

During the past two years the name of this famous obverver has been brought prominently before the public in connection with his views on the artificial prolongation of life, views which have, I may say, particular interest for me, inasmuch as I was, I believe, the first, at all events in this country, to put forward the suggestion that senility is not the result of a mere wearing out of the organs and tissues, but is in large measure self-induced (much in the same way as puberty is) by the coming into operation of special mechanisms provided for the purpose. I conceived, however, that these mechanisms were of a complex nature, such, for instance, as might be provided by specific changes in internal secretions. Metchnikoff's view that senile changes are essentially due to the absorption of poisons from the large bowel appears to me to have little to commend it.

Among the animals in the institute which were being kept for experiment was a juvenile chimpanzee, which was badly suffering from a cold in the head. I have elsewhere expressed the fear that the traffic in the large apes (chimpanzee, orang-outang, and gorilla) for the purpose of experiment may result in their extermination, and apart altogether from sentimental reasons this would be nothing short of a calamity. There can be no doubt that we are descended from a being allied to these creatures, and that the gap between them and us is no longer unbridgable. They offer us inexhaustible opportunities for future research other than by vivisection; and now that we have mapped out their motor cortical centres, now that we can show that they can be syphilised, and that their blood in its reactions exhibits a striking affinity with that of man, I venture to hope that they may be reserved for a happier fate than the scalpel and the injecting syringe.

While in Paris we had the good fortune to meet Professor Babinski, whose name is so well known in connection with the "toe phenomenon." Among his recent works I may refer to his valuable paper entitled "My Conception of Hysteria and Hypnotism," wherein he seeks to establish the essential identity of these two conditions, which, he contends, "are made of the same dough and run into one another." Hypnotism he defines as "a psychic state rendering the subject presenting it susceptible to the suggestion of a second person," and he regards the phenomena which can be induced by suggestion in the hypnotised individual as identical in nature with the functional disturbances observable in hysteria, these suggested phenomena being, both in the hysterical and the hypnotised individual, removable by suggestion, or, as he prefers to put it, by "persuasion." According to Babinski hysteria is, in fact, a peculiar psychic state, the essential feature of which is suggestibility, and its manifestations the result either of auto-suggestion, or of suggestions unconsciously made by the physician. He has satisfied himself that such phenomena as hemi-anæsthesia, contraction of the visual field, and dyschromatopsia are initiated in the latter way. This view is of considerable interest from the therapeutic standpoint. If the state of hypnotism is identical with that of hysteria, it is obvious that the physician should not too readily resort to hypnotism in the treatment of hysteria. Much may, however, be done by "persuasion" without hypnotism. Only in the most obstinate forms of hysterical manifestation does Professor Babinski find it necessary to hypnotise the patient.

At Munich we were fortunate in falling in with Dr. Wrench, a young English physician who gave us much topical information, and through whose kindness we were able to visit the new Anatomical Hall, surely the most magnificent in the world.

* "La Démorphinisation," 'Presse Médicale,' April 23rd, July 6th, 1898.

Dr. Wrench has for several years been engaged in philosophical research, and he is at present staying in the land of the Germans in order to study the customs, institutions, and method of thought of that great people. It will be long before I forget my walk with him under a frosty, starry sky, through the streets of this historic city. I was the more interested in his conversation in that I had just read his recently published book 'The Grammar of Life,'* a work characterised alike by great originality of thought and breadth of view. It is refreshing to be brought into touch with a man who, in the face of great difficulties, uses his commanding abilities in the search after truth rather than in the pursuit of mere material advancement.

Our object in visiting Munich was to inspect the Psychiatric Institute, more especially the Irrenhaus, presided over by the renowned Von Kraepelin. This institution is elaborately equipped with every modern appliance for the investigation and treatment of mental disorders, and is utilised to the utmost for the teaching of psychiatry. We had an opportunity of examining numerous instruments —most of them of Von Kraepelin's own design—for testing psychic phenomena. Two features in the management of the patients at the Irrenhaus may be referred to:

(1) They are not allowed to leave the wards to go to the "Abort," which is placed behind a curtain in the corner of the ward, and are thus not removed from supervision.

(2) Noisy and excitable patients (Die Unruhige) are kept, if necessary for days together, in a warm bath. Here they take their meals, and here they sleep (the head resting during sleep on a support above the water). Professor Von Kraepelin strongly advocates this method of treatment. He more than once drew our attention to the comparative quiet and calm which prevailed in the wards. It so happened that on the occasion of our visit one of the wards was somewhat noisy; but this he assures us was quite unusual, and due to the disturbing influence of one noisy patient, who unfortunately could not be put in the bath because he was suffering from chronic otitis media. Drugs the Professor employs "as little as possible," and recourse to mechanical restraint is never required. Curiously enough, the day after visiting the admirably organised and beneficent Irrenhaus, we saw, in an asylum of a neighbouring country, a young girl squatting in a square cage and yelping like some wild beast. She looked for all the world like a caged ape. It is true that her cage was made of cord; nevertheless the sight was repulsive in the extreme, and carried the mind back to the time when the insane were thought to be literally "possessed" by devils. Nothing could better show the advantage of travel in disseminating knowledge than the experiences of these two days. Here were two cities separated from one another by only a few hundred miles, and yet so far removed in their methods of treating the insane that they might have belonged to different epochs of the world. It was owing to the chance circumstance that we happened to visit the one city directly after the other that the two methods of treatment were brought into such sharp and dramatic contrast.

No city in Europe is more interesting from the medical point of view than Vienna. Naturally we first visited the world-famous Algemeine Krankenhaus. Dr. Frankl-Hochwart, the Professor of Neurology at the Vienna Medical School, conducted us through the neurological department of this ancient building. This vast hospital consists of a group of buildings belonging to a bygone era in medical evolution, and soon to be replaced by a magnificent up-to-date erection in the suburbs, the large centrally situated area of ground on which the old hospital stands being a valuable financial asset. Among the interesting cases which the professor showed us was one of a young woman, suffering from tumour of the brain, probably implicating the pituitary body. Since her illness she had gained considerably in weight, and it was pointed out that many cases had now been recorded of patients suffering from tumour in this region in which the same tendency to put on fat had been observed.

Among Prof. L. v. Frankl-Hochwart's published writings are his exhaustive monographs on tetany in adult life and on Ménière's disease,* both of which are in their second edition and edited up-to-date. They are valuable not only because the professor has himself personally studied a large number of cases of these two affections, but also because he has followed the course of individual

* Published by Heinemann.

* 'Die Tetanie der Erwachsenen Wien und Leipzig,' 1907; 'Der Menièresche Symptomen Komplex Wien,' 1906.

cases during the last two decades. In this way he has been able to show that the prognosis in tetany has hitherto been "much too optimistic." Thus of fifty-five cases whose history he was able to follow over a series of years, eleven had died uncured, and of the remaining forty-four only nine remained healthy. On the other hand, his investigations show that the outlook in Ménière's disease is more hopeful than has generally been thought. He finds that of seventy-four cases of this disease, forty got well, twenty-one improved, while only thirteen showed no improvement. Prof. Frankl-Hochwart was the first to point out that tetany may exist in "*formes frustes*." Patients may merely complain of paræsthesia, examination showing, however, that the muscles and nerves exhibit the characteristic mechanical and electrical excitability. He it was, too, who showed that the best way to induce the Trousseau phenomenon (*i. e.* the induction of spasm in the upper limb by pressure on the brachial plexus) is by pressing upon the bicipital groove.

While at Vienna Professor Van Noorden invited us to attend one of his clinical lectures. Out of consideration for us he chose for his subject diabetes, rightly concluding that we would wish him to discourse on one so closely associated with his name. He began by insisting that the object of dieting the diabetic is not so much to diminish the loss of sugar from the body as to reduce the quantity of this substance in the blood, inasmuch as hyperglycæmia leads to profound nutritive disturbance (such as arterio-sclerosis and peripheral neuritis), which is a much more serious affair than the mere loss of sugar. Although the degree of glycosuria is a fair indication of the degree of glycæmia, it is well to estimate the latter also. Normally it ranges from ·05 to ·07, but in diabetes it may rise to ·4, or even higher. A point of considerable interest is that there is no constant relation between the amount of sugar and the amount of acetone in the blood. Thus, in the subject of the lecture the amount of acetone in the blood steadily fell as the result of dietetic treatment, while the hyperglycæmia long refused to yield. (On the other hand, it must be familiar to many physicians that the breath of the diabetic patient may remain highly acetous, thus showing the presence in abundance of acetone in the blood after the disappearance of glycosuria.)

It was further pointed out that in the treatment of diabetes it may be necessary to limit the proteid intake, and furthermore, that it is by no means a matter of indifference what kinds of proteid are curtailed. Thus the ingestion of casein is especially liable to aggravate glycosuria, while egg albumin and vegetable albumins are much less provocative of it. Bearing in mind that certain proteid molecules, as Pavy was the first to show, contain a carbohydrate moiety, I asked the Professor after the lecture if the aggravating effect of proteids on glycosuria could be explained by reference to this fact, and he replied that he thought not, because casein, which is highly productive of glycosuria, contains no proteid moiety, while egg-albumin, which of all simple proteids is the most rich in carbohydrates, is the least productive of it.

Professor Van Noorden finds that oatmeal is better tolerated by the diabetic than any other form of carbohydrate food, but, before prescribing it, he puts the patient on a rigorous diabetic diet, cutting off carbohydrates, and even diminishing proteids. In this way the ability to assimilate carbohydrates may be increased (Erhebung der Toleranz). A similar good effect, in some cases an even better effect, may be obtained by restricting the carbohydrate food to milk, to rice, or to potatoes; but Van Noorden is convinced of the superiority of the "oat-cure" for the majority. The essential feature of these treatments would appear to be the limitation of the carbohydrate food to one particular kind, and the exclusion or reduction in the amount of meat.

While the diabetic patient is undergoing treatment a careful record should be taken of his weight and strength. If a given dietary causes him to get thinner and weaker it is doing harm even though the glycosuria gets less.

I may here remark that it is not the custom for the students to applaud the lecturer, either in Germany or Austria.

Dr. Porges, one of Professor Van Noorden's assistants, has devised a method of the serum diagnosis of syphilitic patients, which he claims to be an improvement on that of Wassermann.* One to two c.c. of blood are taken from the finger and transferred to a small test-tube. The corpuscles are allowed to sink and the upper serous portion is drawn off. This is then centrifugalised

* 'Wien. klin. Wochenschrift,' xxi, No. 23.

and the serum again drawn off, and heated in a water bath for half an hour to a temperature of 56° C. By means of a finely graduated pipette 2–10 c.c. of the heated serum is transferred to a test-tube (A), and a similar quantity to a second test-tube (B). To test-tube A is added 2–10 c.c. of a freshly prepared solution of sodium glycocholate (1 per cent. of the salt in distilled water), the two fluids being mixed by carefully inverting the tubes a few times, and to test-tube B is added a similar quantity of normal saline solution. The two tubes are kept at the ordinary temperature of the laboratory for about twenty hours. In the event of a positive reaction, at the end of this time a flocculent precipitate will be observed in test-tube A, floating in the upper portion of the fluid, the fluid in test-tube B remaining, on the other hand, quite clear.

It is not maintained that the absence of a reaction (= negative reaction) is proof positive that the patient experimented on has never had syphilis; it is, however, contended that a positive reaction is definite evidence of past syphilitic inoculation: 60–70 per cent. of tabetics give a positive reaction, and 80–90 per cent. of general paralytics. Dr. Porges kindly demonstrated this test to me, and I had the opportunity of witnessing the characteristic flocculent precipitate in one of the laboratories of the Algemeine Krankenhaus twenty hours after the initial processes had been completed. The occurrence of this precipitate was eloquent indeed, for here we had evidence visible to the naked eye that the blood of a syphilised individual many years after the primary inoculation differed from normal blood in every definite way. Dr. Porges maintains that this test is not only simpler and more easy of application than the Wassermann test, but that it gives better results. We found, however, that the two medical residents in charge of the syphilis block at the Stadtische Klinic at Frankfurt placed greater reliance on the Wassermann method. Perhaps it is a case of "a prophet is not without honour save in his own country."

One of the most interesting medical institutions in Vienna is the private sanatorium presided over by Doctor Urbantschitch (the son of the eminent neurologist of that name), who conducted us over it himself and was at great pains to explain its many resources.

The building is situated on an elevated part of the suburbs, and cost £120,000. It is fitted up with every modern appliance for the diagnosis and treatment of disease by non-surgical methods. Not only does it contain clinical and pathological laboratories as well as X-ray and photograph rooms, but it is elaborately equipped with hydro- and electro-therapeutic plant. It is further provided with a room fitted up with numerous Zander machines, a gymnasium, and a work-shop. The Viennese physicians send their patients to this sanatorium, much as we in England send patients to "medical homes." But what a contrast between it and any medical home! We English are in great need of properly equipped sanatoria. No doubt the numerous "surgical homes" in such a city as London provide the surgeon with all that he wants, but of "medical homes" other than those where the patients can be put to bed and nursed, this great city can scarcely be said to have any. Consequently the London physician has not a fair chance of applying all the resources of therapeutics to his private patients; he is obliged to send them for treatment out of London, where they are removed from his personal care. It would be a great advantage if our large cities were provided with sanatoria similar to the one just described, where all the resources of medical science (short of climatic treatment), even to the extent of preparing suitable vaccines, would be available. I believe that a sanatorium of this kind in London, containing, say, a hundred beds, would very soon be refusing applications for admission.

Here I would observe that in the matter of treatment the English doctor differs in an important respect from his continental brother. The former is for the most part content merely to order the treatment, which is carried out either by the patient himself or by others, while the latter not only actively superintends the treatment, but in large measure actually conducts it. He it is who applies the eye ointment or the eye lotion, and who personally undertakes the mechanical (including massage), electrical, and hydro-therapeutical treatment. Though this plan may not altogether commend itself to English ideas, there is much to be said in its favour; for the more closely physician and patient are associated in treatment, the more precise and thorough is it likely to be; moreover, this close personal relation enables the physician to modify his treatment according to need, while it

tends to beget in the patient a confidence in the doctor which makes for success.

I am tempted here to refer to the peculiar position which the pure consultant in this country occupies in regard to treatment. By "pure consultant" I mean the physician who only sees private patients in consultation with another doctor. Now if we assume that the medical man, in order that his therapeutic skill shall have full scope, should, even though he does not actually carry out the treatment personally, at least superintend it, if necessary from day to day, or even from hour to hour; if we assume so much, it is obvious that the purely consulting physician is severely handicapped in the practical exercise of his craft. And in point of fact there can be no doubt that he is. Speaking generally, the most he can do is to arrange a plan of therapeutic campaign with his medical colleague. It is, however, rarely possible to map out beforehand, in its entirety, that stereotyped plan of treatment for a case which shall be the most effective possible: the treatment has to be regulated according to its progress. This is the most unsatisfactory aspect of our present consultation system. The doctor in charge should, as far as possible, keep the consultant and patient in touch during the progress of the treatment, and the institution of sanatoria of the kind referred to would, I think, tend to facilitate the necessary co-operation. A further advantage attaches to the sanatorium. It often happens a patient is sent up from the country to a consultant, and the latter is expected to give a full and final diagnosis after a short interview. But this may be quite impossible; before an opinion can be arrived at it may be necessary to examine the patient in bed, not merely once, but perhaps several times; or again, it may be needful to make daily examination of the blood and the excreta. A sanatorium would enable all this to be done, and thus place the physician in a position to make a diagnosis.

Berlin came next in our itinerary. The most magnificent hospital in this city—perhaps in the world—is that erected as a national memorial to Rudolph Virchow, costing, when fully installed, close upon a million sterling. It consists of fifty-seven buildings, erected on sixty-three acres of ground, and containing two thousand beds. It is built on the pavilion system, the wards being situated on the ground floor, with nothing but the roof above them, but there are underground rooms, or cellars, all of which inter-communicate by passages, which provide conduits for steam, waste water, hot baths, electricity, telephones, etc. The beds include 180 for infectious diseases, 220 for midwifery and gynæcology, 50 for diseases of the ear, throat, and nose, 50 for diseases of the eyes, and 520 for sexual and skin diseases. The only comment I have to make of this splendid hospital is that alike, from an artistic and the practical point of view, it appears to me to be as near perfection as can well be conceived. Doubtless improvements are possible in some of the minor details; otherwise it is probable the designers have reached the high water-mark in hospital construction. The hospital most closely associated with the Medical School of the Berlin University is the Charité. One wing of this building, complete in itself, and to all intents and purposes a special hospital, is devoted to neurology. It is a remarkable fact, however, that nowhere on the continent is there a special hospital for diseases of the nervous system alone, such as that in London for the paralysed and epileptic at Queen Square. The neurological wing of the Charité is under the direction of Prof. Ziehen, who conducted us over it. As might be expected, it is provided with the most modern appliances for the investigation and treatment of nervous disorders, but there is none of that lavishness—I had almost said reckless lavishness—met with in some of the more modern continental hospitals. There are, *e.g.*, only about a dozen Zander machines as against sixty or more at the Workmen's Sanatorium at Bielitz. We had the advantage of attending one of Prof. Ziehen's lectures. The theatre was crowded, and the students listened with great earnestness. The professor is a great man in the University, and is treated with a respect which is not unmixed with awe. He began his discourse directly he entered, spoke rapidly, and was very precise and didactic. He selected for mention the names of two students from a list—those of a young man and a young woman—and their respective owners stood up in front of him during the entire lecture and were subjected to a series of questions after the Socratic method. I am bound to say that the young man looked somewhat sheepish, and did not greatly distinguish himself in his answers. His companion, on the other hand, united a

becoming modesty with no small degree of intelligence.

Among other physicians whom we met at Berlin was Professor Oppenheimer, one of the foremost continental neurologists. Dr. Oppenheimer is not at present attached to the university, but he presides over an important private polyclinic for nervous diseases. The private polyclinic is an institution peculiar to the continent. It is under the control of one man, and may be described as an out-patient department where patients obtain advice, but not medicine. Patients are sent to it by doctors, or come independently for the express purpose of obtaining the opinion of the chief. Dr. Oppenheimer's polyclinic, though lodged in modest quarters, is complete in itself, and is run in a very thorough and practical manner. The professor carefully examines every case, and dictates notes which are taken down by a clinical assistant; he himself examines the eyes, ears, throat, etc., and is assisted in this work by a staff of specialists. There is a clinical laboratory in which examinations of the blood, urine, etc., are undertaken; also a pathological laboratory, X-ray's room, and museum. Patients needing operation are sent to hospitals and sanatoria, where the history of every case is carefully followed, and it is in this way Dr. Oppenheimer is able to obtain morbid tissues for examination. His museum is all the more interesting, in that every one of the numerous macroscopic and microscopic specimens it contains has been prepared either by himself or Madame Oppenheimer. The collection gives evidence of untiring energy, enthusiasm, and patient industry. There were, I may remark, thirteen specimens of tumour of the spinal cord which had been removed on the professor's diagnosis. One half of the patients operated on recovered. There was also a large collection of cerebral tumours; of eighty cases in which Dr. Oppenheimer recommended an operation the majority, he informed me, had died. The professor is an illuminating teacher, very clear, very thorough, very unassuming. His polyclinic is largely attended by medical men, among whom were a few Americans. I may here say that the American doctors I met on the continent impressed me as much by their ability and their earnestness as by their courtesy.

Another of our visits in Berlin was to the private polyclinic of Dr. Cornelius, whose pathological views, if somewhat heterodox, are yet not without interest, and whose method of treatment, though it may not appeal to all, is said to be highly effective. Doctor Cornelius is of opinion that a large number of diseases take their origin in the peripheral nerves, which he believes to be the seat of localised lesions, and that it is to these treatment should be directed. In order to locate the lesions in question the entire surface of the body is carefully explored, by means of the finger-tips, for tender spots, these spots being subsequently indicated on charts. Their existence is rendered evident partly by the patient's wincing, and to a less extent by the occurrence of a localised spasm at the site of the tenderness. The patient attends either thrice weekly or every day, and the tender spots indicated on the chart are successively rubbed with a sort of vibratory movement, each day's treatment lasting on an average about twenty minutes, and the entire course extending over some weeks. The first effect of the treatment is to increase the tenderness in the affected areas; after this the tenderness, it is said, becomes progressively less, and eventually disappears, the associated disorder undergoing a parallel improvement. Though this method is probably most successful in such affections as sciatica and rheumatic troubles, it is also useful in a wide range of disorders. Thus we saw a woman being treated for the vomiting of pregnancy, also a child who was undergoing treatment for chorea. Though he is willing to admit that suggestion may be a part factor in the good results, Dr. Cornelius is convinced that it is not the sole factor, and that the mechanical treatment applied to the tender areas plays the chief part in the successful result. He is assisted in his polyclinic by a large staff of fully qualified doctors, who, clad in white linen overalls, themselves carry out the treatment without the aid of nurses of any kind. This is a feature of the treatment on which great stress is laid. All the work done at the polyclinic is gratuitous, and so great is the demand for treatment that a number of patients have to be refused daily. Dr. Cornelius is so firmly convinced of the efficacy of his method that he is approaching the German Government with the view of getting them to provide the means of placing his treatment within the reach of a larger number of people than the present inadequate buildings and

staff permit. The doctor took great pains in explaining his views and his methods to us, and he is anxious to bring them before the medical profession of this country.

I must not forget to mention that at Bielitz, situated beyond Potsdam, and some thirty miles from Berlin, we paid a visit to a workmen's sanatorium, which is constructed and equipped on an unusually sumptuous scale. In no other place have I seen so many different varieties of Zander machines, and as for the Balneological Block, I doubt if it can be surpassed anywhere. Not only is it provided with every description of douche- and water-baths, but also with hot-air and steam-baths, electric and radiant-heat baths, and even with such luxuries as sand- and mud-baths. In order to show the lavish scale on which the treatment is carried out, I may mention that the earth for the mud-baths comes from a considerable distance, and, though very expensive, is never used more than once, the reason being that the patients would object to such a procedure. The provision of such sumptuous sanatoria has the drawback that the patients are apt to be discontented on returning to their squalid homes. It is, on the other hand, urged that the more thorough and efficient the means adopted for getting the patients well the fewer will there be on the sick list and the less the total cost to the community.

These sanatoria form part of a vast system of insurance against incapacity from age, sickness, or accident. Every workman has to pay out of his earnings 2½d. per week into the general fund, and the employer has to pay a like sum every week for each employee, this amount being over and above the wage as stipulated by the trades union to which the workman belongs. It should be mentioned that those who are disabled by accident draw a special pay, and, as might be expected, malingering is common; and one of the greatest difficulties the medical officers have is to distinguish between real and pretended traumatic neurasthenia.

In Frankfort we had the good fortune to be shown over the neurological wing of the Städtische Klinik by Professor Ludwig Edinger and to examine his valuable museum. This physician, whose researches into the comparative anatomy of the vertebrate brain are so well known, has devised a highly ingenious method of constructing enlarged models of diminutive brains. A series of transverse vertical sections of the hardened brain is taken by means of an instrument which the Professor has himself devised. These are then traced, and re-drawn on a magnified scale on thin cards; the outlines are then cut out, and the cards being placed together in their proper order form an accurate model of the brain, which can then be cast.

In a recently published monograph * Professor Edinger points out how the comparative study of cerebrology (to use a convenient term) and of comparative psychology may help us to discover the functions of the different parts of the brain. He speaks of an "old brain" (palæ-encephalon) and a "new brain" (ne-encephalon). The latter is represented by the cerebral hemispheres; the former by the cerebellum, pons, medulla, and remaining basal structures. It is pointed out that the palæ-encephalon remains fundamentally the same throughout the entire vertebrate kingdom; it is the seat of reflexes and primitive instincts, and although animals with brains consisting of the palæ-encephalon alone are, to some extent, capable of profiting by experience, Edinger argues that there is an essential difference between the psychic activities of the old brain and those of the new. Fish may be regarded as purely palæ-encephalic, though the germ of a cerebrum is to be observed in the shark; in the Amphibia this structure is very definitely represented, in the reptiles still more clearly, while in birds and mammals, especially the higher ones, it attains considerable development. It has hitherto been thought that the only animals possessing a larger brain than man are the elephant and the whale, but Prof. Edinger showed us the brain of a dolphin (concerning whose psychic activities, as he observes, we know next to nothing) of which the cerebral hemispheres present a convoluted surface "unmistakably greater than that presented by man's brain"! (*op. cit.*, p. 21).

The first parts of the palæ-encephalon to develop cortical connections, according to Prof. Edinger, are those which subserve the sense of smell and what he terms the "oral sense." This latter, hitherto almost unstudied sense, is subserved by fibres of the fifth nerve, which are distributed about the mouth and the tongue and are associated in the palæ-encephalon with the lobus par-

* 'Die Beziehungen der vergleichenden Anatomie zur vergleichenden Psychologie,' Leipzig, 1909.

olfactorius, a structure situated just behind the olfactory lobe. Present already in fishes, the lobus par-olfactorius attains considerable development in reptiles, *e.g.*, the chameleon and the snake, and in certain mammals, *e.g.*, the hedgehog, the mole, the pig, and the elephant; but it is small in the ruminants, the monkeys, and in man, in whom vestiges of it are to be found in the anterior perforated space.

The optic nerves appear to have no cortical representation in fish, amphibians, or reptiles. Not until birds and mammals are reached is evidence of a visual cortex to be found.

Prof. Edinger dwells upon our ignorance of the particular functions of the various cortical areas in man. He reminds us that beyond the facts that certain limited portions of the cortex subserve a motor function and that other limited portions subserve the functions of vision, we know little or nothing of the functional topography of the cortex. I gathered from him that he regards the frontal lobes as occupying the highest place in the cerebral hierarchy.

Human brains are sent to Prof. Edinger's laboratory from all parts of Germany in order to obtain his expert opinion regarding them. For this purpose special boxes containing all the necessary appliances for hardening and packing have been constructed, and some twenty of these are in constant circulation.

Perhaps Prof. Edinger is best known in England in connection with his views concerning the influence of functional activity in the production of organic affections of the nervous system, more especially those in which toxicity is thought to play a prominent part. The professor's first pronouncement on this question was made some fourteen years ago. Since then he has been carefully testing the views he then advanced, with the result that he is more than ever convinced of their soundness. In an exhaustive monograph recently published* he reviews the whole subject. He argues that the changes which take place in a normally constituted neuron as the result of its normal functional activity are of the same nature as those which usher in degeneration, and that when this functional activity is excessive actual degeneration may ensue. Thus he has shown that when a rat is strung up for some time by the tail the continued struggles of the creature cause such exhaustion of certain afferent neurons as to lead to degeneration of the posterior columns of the cord. This fact—that excessive functional activity can set up degeneration in a normal set of neurons—prepares us for the conclusion that moderate functional activity may be capable of initiating degeneration in defectively constituted neurons whether that defect is congenital or acquired. In Friedreich's disease, for instance, we may suppose that certain tracts in the cord are congenitally defective, the spastic ataxic disturbances characteristic of this affection only developing as the child comes to use his legs actively in walking. Similarly it is held that in progressive muscular atrophy and progressive bulbar paralysis there is a congenital defect of less pronounced degree in certain of the lower neurons, which only begin to degenerate after many years of functional activity. Among the conditions which set up acquired weakness are mentioned—toxæmias (*e.g.* from lead, arsenic, and alcohol), prolonged dyscrasias (*e.g.* cancer, tuberculosis), anæmias (from atheroma), and infectious diseases. It is well known that under any of these conditions changes which are attributed to "neuritis" may occur in the peripheral nerves, and it is argued that in the production of such changes functional activity may play an important part, so that if the weakened neurons are kept at rest it may be possible to hold the affection in abeyance, or if not this at least to minimise its severity. It is not, of course, denied that degeneration of certain neurons may occur under the completest possible physiological rest (*e.g.*, in diphtheria); that, however, "functional activity" may determine the degeneration in neurons of which the vitality has been artificially enfeebled is shown by Edinger's remarkable experiments with pyrodon. He finds that if rats to which this substance has been given are kept perfectly quiet, no degeneration takes place in the nervous system, but that if they are allowed even a moderate amount of exercise, degeneration of the posterior column of the cord takes place—in other words, moderate functional activity of weakened neurons is able to set up exactly the same kind of degeneration as excessive functional activity of normal neurons.

Prof. Edinger contends that in most cases of so-called toxic degeneration of the nervous

* 'Der Anteil der Funktion an der Entstehung von Nervenkrankheiten,' Wiesbaden, 1908.

system, the functional factor plays an important part both in precipitating the lesion and in determining its site. Thus the reason why the extensors of the wrist are so readily affected in lead-palsy is because in using the paint-brush the chief muscular stress falls upon these muscles, and when it is reflected that they may be called upon to contract as many as 2800 times in the hour, it will readily be understood how great that stress must be. It is of interest to note that the supinator longus and the long extensor of the thumb-muscles, which are very little used in painting, usually escape paralysis.

Prof. Edinger gives many instances showing the influence of the functional factor in the pathology of tabes dorsalis. A few of these may be cited here. The patients referred to were the subjects of genuine tabes. A man, after hunting all day in a district covered with snow, exhibited dilated pupils which refused to contract to light; another, who had been obliged to sit up watching all night, and had experienced the greatest difficulty in keeping his eyes open, showed marked ptosis the next day; a third, whose business it was to test the freshness of several thousands of eggs every day by holding them up to the light, developed optic atrophy. Pointing in the same direction is the fact that ataxy develops in patients who exercise their legs strenuously more frequently than among the sedentary and those whose activities are curtailed by the early onset of optic atrophy, or than among the comparatively inert peoples of the East.

It is scarcely necessary to point out that a due recognition of the part which the functional factor plays in the ætiology of the degenerative diseases of the nervous system is of considerable importance therapeutically. Thus Prof. Edinger advises his tabetic patients to wear coloured glasses, he insists that the bladder shall be emptied every two hours, and he prohibits them from making undue exertion of the muscles engaged in maintaining the bodily equilibrium.

It was interesting to find that Conan Doyle's works are largely read in Germany and Austria, where the mere mention of his name was sufficient to arouse enthusiasm. It did not seem to be generally known that he is a member of our own profession. His new books are eagerly looked forward to both by parents and children. Prof. Van Noorden provides himself with his latest story when he has a long journey to make; Professor Edinger takes up one of his volumes in the intervals between his investigations on the brain; while Professor Ehrlich finds solace among his pages amid his laborious researches into the chemical mysteries of the blood.

May 2nd, 1910.

ON TWO COMMON ERRORS IN THE ADMINISTRATION OF ANÆSTHETICS.*

By J. D. MORTIMER, M.B., F.R.C.S.,
Anæsthetist, Royal Waterloo Hospital, Throat Hospital, Golden Square, etc.

If I were asked, "What are the two most common errors of the unskilled anæsthetist?" I should reply, "A failure to appreciate the importance of choosing an anæsthetic suitable to the patient and to the operation; and a failure to appreciate the importance of maintaining a free air-way."

The choice of an anæsthetic.—I am sometimes asked by practitioners, in the operating theatre, or on my arrival at a private case, such a question as—"Do you generally give ether or chloroform?" "What anæsthetic are you going to give?" And when I reply, "I do not give either as a matter of routine," or "I do not know until I have seen the patient," such an answer is evidently unexpected.

Now it is in the ability to select an anæsthetic with due regard to all the circumstances, and in the ability to administer any kind of anæsthetic and to change from one to another, as may prove desirable, that a good anæsthetist's skill and experience is made evident; on the other hand, such inquiries as I have cited indicate that the questioner's knowledge of the subject is not extensive.

It need hardly be said that the welfare of the patient should be the main consideration. This, however, is often regarded in too limited an aspect—almost as if the administrator may be considered to have done his duty if he has escaped an immediate fatality! Suppose, for example, that a stout, emphysematous woman needs a prolonged ab-

* Being opening remarks at the Special Class in Anæsthetics, Medical Graduates' College and Polyclinic.

dominal operation. Ether will probably be given in a Clover's inhaler, on the ground of being "so safe," if the anæsthetist be unskilled, with likely results of considerable discomfort to the patient, delay and difficulties in the operation, and grave complications afterwards.

The question must be regarded not only from the point of view of *the immediate safety of the patient*, but also from the following: *the experience of the administrator; the patient's age, physique, and special conditions of health; the nature and probable duration of the operation, and any special requirements of the surgeon.*

It will be found in practice that these factors to some extent overlap, and that one cannot attend to one to the exclusion of the others: "Every case of anæsthesia is a study in itself" (Marmaduke Sheild).

In dealing with anæsthetics in common use, with varieties of patients, and with anæsthetisation for certain operations—abdominal, renal, nasal, etc., —we shall discuss all these points except the second, and I believe you will agree with me eventually that my opening remarks are justifiable, even if you may not yet be inclined to do so.

As regards the *experience of the anæsthetist*, if he is fairly conversant with some method of using one anæsthetic and its effects upon patients rather than with another, this may turn the scale when doubt exists. If he is altogether inexperienced he should certainly choose that which as a rule is the safer, *i.e.* N_2O rather than ethyl-chloride, ether rather than CE, and CE rather than chloroform. But it is only in emergencies such as occur in country practice that such a one should undertake the task at all, particularly when the other factors to which I have alluded are more preponderant than in a simpler case. A practitioner working under ordinary conditions should not be expected—and should not himself expect—to be a specialist in anæsthetising any more than in operating.

The maintenance of a free air-way and attention to the respiration.—I wish to draw your attention particularly to this, because there seems to be an impression prevailing that fatalities, especially under chloroform, are due usually to *primary* failure of the heart, whereas in many, if not in most instances, such failure is either *secondary* to *respiratory obstruction*, or is *conjoined* with gradual *respiratory failure*, the signs of which have not been duly appreciated. It may be said—"Why not use one word—asphyxia—to denote these conditions?" Because the word, from its application to cases of suffocation, such as by smoke or from choking, is apt to bring before the mind a clinical picture which differs widely from the condition produced by want of oxygenation in a person under the influence of most anæsthetics. In the latter the cardinal signs met with in the former, such as congestion and cyanosis of the face, convulsive movements and so forth, are often so lessened and postponed by the effect of the anæsthetic upon the nervous centres that an administrator who trusts to them as danger-signals may find himself unexpectedly in a very anxious position. In failure especially there may be nothing but slight lividity and somewhat gasping, irregular, or shallow breathing when the function is on the point of extinction. One must not forget that although the patient takes in less anæsthetic when breathing badly he also fails to eliminate it.

Obstruction of the respiration, although less serious than *failure*, is injurious to the patient not only from retention of gaseous excreta but also from the difficulties created in the circulation of the blood. Free respiratory movements not only fill the lungs with air, but are also a powerful factor in filling and emptying the pulmonary vessels and drawing blood from the great systemic veins into the heart. When they are hampered the heart is not only affected by the impurity of the blood supplied to itself, but may become so dilated mechanically that it fails to contract. This is especially apt to occur in mitral stenosis or if previous degeneration exist, as in the flabby heart of beer-drinkers ("Münich heart"). Even under N_2O such conditions may be very dangerous.

The effects of respiratory obstruction are also very annoying to the operator. Venous oozing obscures his view and in some situations may lead to disaster. There may be undue rigidity or spasmodic excursions of the diaphragm and abdominal muscles which make the operation difficult or even impossible.

It has also been shown that after-sickness is more likely to occur.

[The causes of respiratory obstruction and failure, and the methods of dealing with them, were then enumerated in detail.]

May 2nd, 1910.

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CONTENTS.

PAGE

*A Clinical Lecture on Chronic Ascites with Cirrhosis of the Liver. By W. P. HERRINGHAM M.D., F.R.C.P. ... 65

*Sympathetic Ophthalmia. By ARTHUR ORMOND, F.R.C.S. 70

*Chronic Malignant Endocarditis simulating Splenic Anæmia ... 73

*Hypernephroma 76

*Bronchiolectasis 79

* *Specially reported for the Clinical Journal. Revised by the Author.*

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A CLINICAL LECTURE

ON

CHRONIC ASCITES WITH CIRRHOSIS OF THE LIVER.

Delivered at St. Bartholomew's Hospital.

By W. P. HERRINGHAM, M.D., F.R.C.P., Physician to the Hospital.

GENTLEMEN,—I want to speak to you to-day about cases of chronic ascites with cirrhosis of the liver, and I use the word "with" instead of "due to" cirrhosis of the liver, because that is exactly the point in dispute. You will find, as I go on, that it is disputed whether certain of these cases about which I shall speak to you are due to, or are merely coincident with cirrhosis of the liver. This man whom you see here is æt. 44 years, a glass-cutter by trade, who came into Colston Ward on October 5th last year. There was a history of his having taken rather too much beer. There was no history or sign of syphilis. He had been in another ward some few months before—I think it was in March of that year—with the same symptoms for which he came to us, namely, swelling of the abdomen, inability to walk, and swellings of the legs. He had then been tapped several times. He had had no previous illness. When he came in he was a well-nourished and fairly healthy-looking man, and he had, I specially wish you to notice, not a cirrhotic facies, that is, a pale face with dilated veins on the cheeks. He has slight cardiac valvular disease. He also had from the first, and has had ever since, ascites. His liver was one and a half inches below his ribs; it was hard and the edge was decidedly thick, but the surface was smooth, so far as we could tell. And I may mention here that I should always advise you to be very chary of trusting to feeling the granular surface on the liver through the skin. It is exceedingly difficult to feel anything like the small

granulations upon the liver. Of course you can feel the bosses of cancer, and you can of course feel the irregularities due to large gummatous syphilitic disease, and sometimes you can feel the irregularities of simple cirrhosis when they are very large, but not as a rule, and what is generally thought to be the rough surface of the liver is the granular tissue in the abdominal wall. That man came in with an abdomen that was 47 in. round, and when he was admitted he passed urine in fair quantity. We put him at once upon saltless diet, which has been found useful in these cases, and for the first week it seemed to increase the urinary outflow; he passed an average of 56 oz. every day, and his girth fell 3 in. In the second week it was not so successful: his average fell to 40 oz. of urine a day, and his girth remained the same. And so I changed his drugs, putting him on citrate of caffeine and infusion of digitalis, and that gave him a certain stimulation of secretory functions. He passed 52 oz. *per diem* for the next week and the week after that, and after that it fell to 32 oz. So I tried something further, and we gave some spirits of juniper and infusion of broom. That did no good at all, and the next two weeks' urine was only 28 and 32 oz. *per diem*. The girth was 43 in. at the end of that time. I put him upon diuretin, and that did him no good; the girth rose in the fortnight that he took it, and he measured 45 in. round the chest at the end of the time. Then, as he was feeling fairly well, I gave him some more to eat; I gave him a steak, with the result of increasing his urine. There were no changes in his habits except for the eating of that steak for his dinner, and he passed 56 oz. of urine *per diem* for the next week. And since that time he was put on the ordinary standard half-diet of the hospital with meat, and he has passed a fair amount of urine, and his girth has gone down to 40 in. We never felt we were obliged to tap, and he refused any suggestion of operation. He seemed to have the power of absorbing some of his fluid in his abdomen, and therefore I suppose there must have been a collateral circulation established or being established, for his girth has sunk from 47 to 40 in. during the time he has been in the hospital, that that collateral circulation is increasing, and that he is getting more and more able to dispose of the fluid in his abdomen. Apart from that he seems to be in fairly good condition. He does not look like a cachectic man, as I think you will agree, he has never been jaundiced, the liver has not increased or diminished in size, and it has exactly the same feeling as when he first came in. I have given him a support in the shape of a belt, the one which you see before you, and what you see in the middle is a pad for his umbilical hernia, which is a separate disease. He is going out of the hospital, and I think he will be able to carry on his employment.

Another case, very like that, was in Colston Ward from July 1st, 1902, to June 13th, 1903, that is to say, almost seven years ago. He was a man called B—, æt. 37 years, a messenger. There was a strong history of alcoholic drink, both beer and spirits. There was no history or sign of syphilis. For a year before he came in he had had nausea and morning vomiting. Six months before he had had severe hæmatemesis, and for four weeks he had noticed his abdomen swelling. On admission he was a fairly healthy-looking man and well covered. His tongue was clean, heart and lungs natural. He had ascites, and his liver was the same size as this man's, one and a half inches below the ribs and heart, and the spleen was not definitely felt. He had a much more severe ascites than this man whom you have just seen. And between July 1st, 1902, and March 3rd, 1903, he was tapped nineteen times. In the total he lost 328 pints of ascitic fluid. In December, January and February, the intervals between the tappings were only eight or nine days, and he lost eighteen or twenty pints each time, so he was filling up then at the rate of two pints a day. The liver remained the same size throughout, and at the end of February the intervals began to grow greater, and after March 31st he was never tapped again. He remained in three months longer, became better and better, and went out wearing a belt very much the same as that man has on, for a support. He still had a swagging abdomen with some fluid in it, but was able to go to work, and he has remained at work ever since. I heard about him only a week or two ago from the sister, and he is still in good health. In both those cases the urine was perfectly natural. You will easily understand that there are many cases of ascites and a large liver which are not cirrhosis at all. For instance, we had a woman in a year or two back who was tapped repeatedly, and she had a large liver and recurrent ascites. In her case it

was due to some disease of the heart, and I have no doubt it was one of those cases of exocardial adhesions which have been called mediastinitis, and which are often accompanied by serous effusions in more than one cavity. But I think there can be no doubt both the cases I am now bringing before you had cirrhosis of the liver of a certain kind. And I show you them and tell you about them because such cases of chronic ascites which are repeatedly tapped are very uncommon. At Guy's Hospital the teaching is very dogmatic about ascites due to cirrhosis, but remember that is not the title of this lecture. Ascites due to cirrhosis is said at Guy's never to survive one tapping. Here is a very interesting paper in the 'Guy's Hospital Reports,' vol. xlix, 1892. I will read you what Dr. Hale White says: "So true is this that I have not, in consulting the records of Guy's Hospital for the past five years, been able to find a single case of uncomplicated cirrhosis of the liver with ascites which has long survived paracentesis of the abdomen; not one has lived long enough for that operation to become necessary a second time, and many have died before it became necessary at all." That is extremely dogmatic. Practically they say at Guy's no case of ascites due to cirrhosis of the liver survives more than one tapping. And the writer goes on: "Whereas cases which are repeatedly tapped are due to a general peritonitis, which usually includes complete peri-hepatitis and perisplenitis, and is almost always coincident with chronic nephritis." Their idea at Guy's is that chronic ascites repeatedly tapped is not due to, even though it may coincide with, cirrhosis of the liver, but is due to chronic peritonitis, and that is almost always coincident with chronic nephritis. I had a very good example of that some years ago. A man, æt. 51 years, a chemist, was admitted on October 11th, 1904, and stayed in the hospital for about six months. He was tapped seven times, and went out, but was readmitted June 21st, and died July 9th, 1905. And then we found, what we knew beforehand, that he had chronic interstitial nephritis, besides the cirrhosis of the liver and a general peritonitis with complete thickening of the capsule of the liver or spleen. His kidneys were 5 and 6 oz. in weight, so that they were not diminished in size, they were the subjects of chronic interstitial nephritis, and the heart did not appear to have been affected. It was 10½ oz. in weight and is described as natural. It was recurrent ascites, of the kind which Dr. Hale White described. And the paper in which he first ventilated this subject is the 'Clinical Society's Transactions' 1888, vol. xxi, in which he gives a case of peri-hepatitis, with an analysis of forty cases, showing that complete peri-hepatitis is nearly always part and parcel of a general peritonitis, and that is coincident with chronic renal disease. That teaching you will find also in Fagge's 'System,' which is again a book of the Guy's school, and of course anything which comes from Guy's is of the greatest authority. I am certain, however, that that dictum is either exaggerated, or else is not of any very great clinical importance. In other words, I am sure there are many more cases than that, which in life you must diagnose as cases of cirrhosis of the liver, pure and simple alcoholic cirrhosis of the liver, in which the ascites is due to the cirrhosis of the liver to all appearances, which yet do not follow such a rapid course as is there described. That is not only my opinion, it is the opinion also of many other people. You will find here in the 'Medical Society's Transactions,' vol. xv, 1892, a very interesting paper by Dr. Bristowe, who used to be the great authority when I was your age. His book on medicine was the one we always read, just as Osler's is probably the one which you read most now. Bristowe was a St. Thomas's man, and his paper on "Observations on the Cure and Subsidence of an Ascites due to Hepatic Disease" in this volume is entirely devoted to that subject. He gives three or four cases which are extremely interesting to read, but which are subject to this drawback, that they are none of them cases on which a post-mortem was made —they all recovered. Dr. Cheadle* published the Lumleian Lectures delivered before the College of Physicians in 1902 in this book, on "Some Cirrhoses of the Liver," and he gives cases in which there was a post-mortem examination, and I will read one or two of them to you. The course of some of the more favourable cases of the atrophic cirrhosis which survive for a time is exemplified by that of a woman who died of intercurrent bronchitis before the liver was so extensively

* Dr. Cheadle died very shortly after this lecture was delivered.

injured as to seriously impair her general nutrition. She was æt. 53 years, admitted for ascites and hæmatemesis. She was known to be a steady drunkard. She was the wife of one of the officials of the hospital school, and he also was a steady drunkard. He was a messenger belonging to the school. She was tapped twice, and nine months later was re-admitted and was tapped again. At that time she had acute pneumonia, and got well of that. A month later she was again admitted, with bronchitis, and this time she died. This was nearly a year from her first admission, and she was tapped three times. The post-mortem examination showed a contracted, typical hob-nailed cirrhotic liver weighing 38 oz., but no general peri-hepatitis or peritonitis. The kidney showed commencing granulation, but was not much contracted. That is one case. The second one was a man, æt. 43 years, a cabman, of intemperate habits. He was tapped nineteen times. He was admitted April 4th, 1904, and sent out June 18th, 1905. He was admitted the following August for hæmatemesis, and died the following day. At the post-mortem examination the liver was found to be contracted, covered with a thick false membrane; the kidneys were normal and there was no general peritonitis. There is a third case of the same kind recorded. That little book is a very interesting set of lectures, and is worth looking at too. I have had, in the last few years in Colston Ward, seventeen cases of cirrhosis of the liver. Eight of them have died in a few weeks, which is the most frequent thing to happen after ascites supervenes. I quite allow that. One of them was tapped six times before he died; eight of them were discharged from the wards. Four of them had no ascites, so they did not come into the present category. Four of them had to be tapped, and certainly four of those who were tapped were in my opinion of that kind, although I cannot say for certain because I have not had a post-mortem on them; they were discharged from the wards. To the best of my knowledge they were cases of cirrhosis of the liver. But you must remember that when you do not get a post-mortem examination on these cases you can never be sure, because in the first place there may be syphilitic disease of the liver. That is not very uncommon, though it is not very common. And in any case of doubt it may always be said your case was not one of alcoholic cirrhosis, or at any rate not alcoholic cirrhosis pure and simple, but of syphilitic cirrhosis. Many people drink and have had syphilis, so it may have been both. And then it may be said that some of these cases had chronic renal disease, and although one thought the ascites was due to the cirrhosis of the liver, yet it may have been due to the renal disease, and have been a chronic general peritonitis due to that rather than due to the cirrhosis of the liver. But still, from the clinical standpoint, if you go into practice you will want to know what is the prognosis in a case of ascites, which you think, after considering it as best you can, is due to cirrhosis of the liver. And as a matter of fact it is not so unfavourable as you may at first be led to think. A certain number of cases which we diagnose very carefully to be cirrhosis of the liver do recover sufficiently to be discharged from this hospital. One of those cases did come to a post-mortem examination, because after tapping some seven times he was advised to have an operation done, what is generally spoken of as the Drummond Robinson operation for producing anastomosis. The surface of the liver is roughened and is stitched to the surface of the diaphragm, which is also rubbed, so that adhesions are formed between the two, and through those adhesions anastomosis takes place, with the ultimate result, in some cases, of relieving the pressure in the peritoneum and providing a way for the fluid to escape. This man had this operation and in a few days he died. It is an operation which is not very successful; many of the cases so treated die. We were able to find that he had a cirrhotic contracted liver and general ascites, that he had recent peritonitis, and that is what he died of. But apparently he had no old peritonitis. He also had hypertrophied and granular kidneys. So there again you see the difficulty of establishing, even when you get a post-mortem examination, sometimes with certainty whether the chronic ascites is due to cirrhosis of the liver or due to the complication of renal disease which exists in some cases.

In some cases you will come across a very curious form of ascites, in which the ascitic fluid is not clear as it is in nearly all the cases, but milky or opalescent. Those form a group by themselves, which are called chylous or chyliform ascites; it is not a matter apparently of great

clinical importance. The interest is to know what makes that peculiar appearance. Dr. Robert Hutchison, in the 'Transactions of the Pathological Society of London,' vol. liii, has shown that some of them, at any rate, are due to a peculiar change in the albumen which is excreted, which the fluid contains, a change which he thinks to be due to some form of nuclein or para-nuclein containing proteid, something derived from cellular disintegration, albuminous degeneration. There is another paper in the 'Quarterly Medical Journal' for April this year, written by one of our own men, which is the best paper hitherto written on the subject.

The treatment of these cases of chronic ascites is exceedingly unsatisfactory. To get rid of the ascites is very difficult. You saw I gave that man all kinds of diuretics, and it is far the best way to get rid of chronic effusion in the abdomen. It is not safe to give these people diarrhœa, and that is what you frequently do if you try to get rid of the fluid by means of the intestines. And yet to get rid of it through the kidneys is exceedingly uncertain and difficult. I think I should say on the whole, having tried it very many times, that you are practically dependent on chance. The kidneys do not respond in these cases to any diuretics that I have ever tried. On the other hand, I have several times known the kidneys take spontaneously to excreting a large quantity of fluid of the ascitic effusion. But the effect of drugs is exceedingly uncertain, and in my experience exceedingly small. You have to tap the abdomen in the great majority of cases. Not in a case such as the man lying here, in whom apparently the collateral circulation is being re-established and the ascitic fluid is being absorbed by the man himself, but in cases where that does not happen, where the abdomen gets very large and the patient has much discomfort. And it is well not to put off the operation too long, because, as in a case of pleural effusion, it is very frequently found that the removal of fluid helps the future removal of the fluid which collects a second time. The relief of pressure apparently enables the serous surface to remove what fluid is left. The best way to do tapping is through a very small tube, we do it through a Southey's trocar, and run it out under aseptic precautions to the side of the bed. But the prognosis in these cases does not depend upon the ascites. The presence of ascites is a very serious symptom, but it is not in itself the cause of death. The cause of death in these cases is toxæmia—there is no other word for it, although I do not know what the meaning of it is—the toxæmia which the liver disease produces, kills the patient, and the patient will die from that by general gradual asthenia, or by that state which we call cholæmia, a state which is very like the condition called uræmia produced by chronic disease of the kidneys. In both there is vomiting, diarrhœa, wasting, anæmia, and cardiac failure, and both of them end in nerve symptoms, delirium, convulsions, and coma. Of course we do not believe those conditions are due to the same poison, but I think they must be due to some analogous poisons, because in both there is the same sort of fibrotic change going on, and there is undoubtedly destruction of cell-tissue. I always have the opinion that it is some poison derived from dying or destroyed epithelial cells. For just as in nephritis you cannot explain uræmia by retention of any known products, so in hepatic cirrhosis you cannot explain cholæmia by the retention of any products that we know of, of hepatic action. The two chief functions of the liver are the production of urea from the products of muscular digestion, apparently through lactate of ammonia, and secondly the stoppage of the carbohydrate portions of the food on their way to the circulation, preventing sugar entering the circulation, at any rate in large quantities. Neither of those effects or functions can be traced with any great certainty in cirrhosis of the liver. It is true the urea is diminished; some say it is diminished towards the latter end of life, and that uric acid is increased, and ammonia is also increased. But that does not happen at all events within three or four months of death, as I can show you from a case we had last year. A man, called W—, died in October of cirrhosis of the liver with ascites. In June we estimated his urinary excretion for ten days consecutively, and he passed on the average 21 grm. of urea, and that is a full proportion for a man living on the ordinary half diet of the hospital. He passed 21 grm. of urea *per diem*, and his ammonia was estimated twice. The last estimation showed only 5 per cent. of ammonia out of the total of nitrogen. So he was not excreting any extra amount of ammonia, but he had his full proportion of urea excretion. And that reminds one again of the cases of chronic nephritis in which you cannot trace the uræmic symptoms to any retention of the ordinary products of the kidney.

May 9th, 1910.

SYMPATHETIC OPHTHALMIA.

A Clinical Lecture delivered at Guy's Hospital.

By ARTHUR ORMOND, F.R.C.S.,
Ophthalmic Surgeon to Guy's Hospital.

SYMPATHETIC ophthalmia is not as common a disease to-day as it was thirty or forty years ago; nevertheless it is still as interesting both to the clinician and to the pathologist as it ever was. To-day I want to interest you in a typical case.

The patient is a man, æt. 65 years, living in Essex and earning his livelihood as a farm labourer; at the end of May, 1909, he was struck a very forcible blow on his left eye with a piece of wood. After the initial pain and discomfort had passed off he experienced no trouble from it, although he seems to have been aware that the sight had been seriously interfered with. Three or four weeks later he was compelled to consult a doctor, finding that the sight of his right eye was failing and he could no longer see to do his work. He came to Guy's

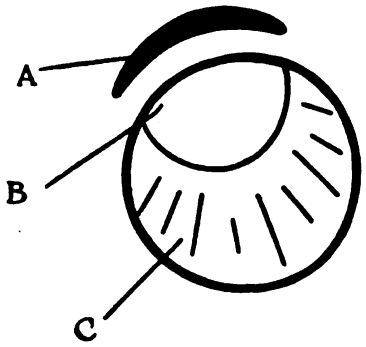

Diagram of left eye. A. Pigmented area in ciliary region. B. Coloboma of iris. C. Stretched iris.

Hospital and I examined him on July 14th. His ocular condition was then as follows: The vision of the left eye was equal to counting fingers at two feet; there appeared to be a quadrant of his iris missing above and to the inner side, giving an appearance similar to that produced by an iridectomy; beyond this gap, at a distance of 4 to 6 mm. from the corneo-scleral margin, and corresponding in width to the coloboma of his iris, was a dark brown pigmented area. The iris was discoloured and stretched quite flat across the pupil and the gap in the iris was filled in by an opaque membrane; the lens could not be seen, but it was evidently not in position. On the posterior surface of the cornea at the lower part numerous dark brown dots were visible (keratitis punctata), and there was circum-corneal injection. The blow this man had received six to eight weeks before had ruptured his eyeball, and he now had a plastic inflammation of the uvea.

The right eye could see $\frac{6}{36}$, having a visual acuity of about one sixth of the normal; there was ciliary injection, some photophobia, and lachrymation. The iris was discoloured, and numerous synechiæ could be seen, also keratitis punctata. The lens was partially opaque. It was obvious that the right eye was also suffering from a plastic inflammation of the uveal tract, and this inflammation had followed a plastic inflammation in the injured left eye.

The patient was admitted to the ward, put to bed, and both eyes bandaged. Mercury was given internally and also as inunction, following hot baths. He improved slightly for two to three weeks, then steadily got worse, and subconjunctival injections of cyanide of mercury and acoin were tried, also dionine, but without any beneficial effect on his right eye.

In November last, however, it was noticed that the left eye, that is, the one that was originally injured, was less red and injected, and the patient stated that he could see better with it.

This improvement was maintained, and in January I decided to break the rule never to operate in cases of sympathetic ophthalmia, and I needled the web in the pupil of his left eye, making a small, nearly central hole through it. This gave a most gratifying result, as the gap in the web remained unfilled in, and with glasses (+12 D. sph.) the man is able to see to go about by himself. The vision of his right eye, the "sympathising" eye, is now only perception of light, and the tension is sub-normal.

The old view of sympathetic ophthalmia was that it was a plastic inflammation of the uveal tract of one eye, following a traumatic inflammation of the same parts of the other eye. This definition, then, implies four conditions: (1) An injury, followed by (2) a plastic inflammation; (3) a lapse of time; and (4) a plastic inflammation in the sympathising eye.

The injury was always represented as being a perforating wound, and more likely to lead to sympathetic trouble if it involved the corneo-scleral margin, which margin for this reason became known

as "the dangerous area," an area which stretched from the corneo-scleral margin backwards for about 6 mm. My experience, however, does not confirm this. Wounds in the "dangerous area" are not necessarily followed by sympathetic ophthalmia, whereas perforating wounds elsewhere may be.

Later on, cases were recorded of this disease in which the wound was subconjunctival, as, indeed, might well have been in this case which we are now considering, and still more recently a large number of instances have been reported in which sympathetic trouble has been found in cases of intra-ocular sarcoma in which anything in the nature of a penetrating wound can be absolutely excluded. Consequently we must, according to modern views, alter our definition of sympathetic ophthalmia, and we must look upon it as a specific disease of the uveal tract, the result of an endogenous infection by ultra-visible organisms. I shall, however, return to this point when discussing the pathogenesis of sympathetic ophthalmia.

Let us now consider the symptoms. The eye which receives the injury is spoken of as the "exciting" eye. The tendency for the eye to recover from the effect of the injury must be carefully remembered; the redness should be less, the pain subside, and the vision improve during the subsequent ten to fourteen days; but in watching a case in which a possible risk of sympathetic trouble is involved, any delay in healing, any increase of the signs of inflammation taking place, say, ten days or a fortnight after the injury, must be looked upon as indications of serious trouble. Consequently the originally injured eye may give you the first sign that all is not going well; further, if you can find evidence of a plastic iritis, such as keratitis punctata in the exciting eye, the eye should be at once removed. Unfortunately, however, the symptoms may be, and often are, very insidious.

The "sympathising" eye must also be carefully watched, as it will give you indications of the onset of this dangerous condition; if there are present in it the symptoms of failure of the accommodation, leading to a mistiness of vision and inability to see print or small objects for any length of time, lachrymation, circumcorneal injection, and haziness of media, it will be equally imperative to remove the damaged eye at once. If this is not done the time will soon come—generally in a day or two—when there will appear incontrovertible evidence of a definite inflammation in the "sympathising" eye (that is, the eye which has not received the injury), and then it is too late to interfere; the removal of the injured eye at this stage will not save the "sympathising" eye, as the iris, ciliary body and choroid will become more inflamed, and subsequently atrophy will ensue, leading to hopeless blindness. The "sympathising" eye, if it escapes a glaucomatous condition, often shrinks up, and, as in our case, the originally "exciting" eye is the eye with the better vision.

I often notice in cases of sympathetic trouble that there are evidences of constitutional disorder, pallor, anæmia, malaise, anorexia, constipation, etc. Actual inflammation does not always follow the early irritative symptoms; sometimes we find evidence that the photophobia and lachrymation of the sympathising eye have been present for a long period without producing any evidence of inflammation. Perforating wounds of one eye may have other results than sympathetic inflammation, such as (1) sympathetic irritation, (2) serous (not plastic) iritis, and (3) neuro-papillitis. Of the first I have an instance at present in the ward—a woman whose eye has been ruptured by a blow from her husband's fist. Of the second I had a case a short time ago: a little boy had a small, fine synechia appear in one eye following two months after a perforating wound to his other eye. The exciting eye was removed, and the synechia disappeared under atropine without any further trouble. The iritis was not of a plastic nature.

Another case which I recorded in the 'Transactions of the Ophthalmological Society' was one where a papillitis remained for eight to ten months after enucleation had been performed for a perforating wound.

The usual time for the onset of the symptoms of sympathetic ophthalmia to appear in the sympathising eye is four to six weeks. No undoubted case of infection of the second eye has been recorded as occurring in less than eleven days, but on the other hand the time that may elapse between the original trouble and the onset of the sympathising trouble is quite indefinite, and may be years.

Pathogenesis.—For years ophthalmic surgeons have been considering the possible method by which the inflammation of the one eye is conveyed to the other eye. You will find in your text-books

pages and pages written on this subject, all of which are inconclusive. We must, however, in considering the facts before us, acknowledge that we are not quite certain how the originally "exciting" eye is infected, if, indeed, it is infected by micro-organisms at all.

In cases of sympathetic ophthalmia associated with intra-ocular sarcoma, it is quite clear that the organisms are not introduced from outside, as there is no wound, and therefore the infection must be endogenous and not ectogenous. Sympathetic ophthalmia is a specific disease in the same way as malaria is a specific disease, and the pathological changes found in the eye are distinct, the plastic masses consisting of lymphocytes, epithelioid cells, and giant cells. It resembles tubercle but differs from it in the absence of caseation, definite tubercle systems, and the tubercle bacilli; these histological changes are similar in the two eyes, and are always found in definite cases of sympathetic ophthalmia, and have also been found in cases of intra-ocular sarcoma. This fact is common to all cases of sympathetic ophthalmia, namely, that some damage has been done, either by injury or disease, to the cells of the uvea, and it is suggested that the ætiological factor can only act on damaged uveal tissue. With regard to a specific organism, up to the present time it has not been isolated. In the case before us I removed some of the aqueous humour, but this proved quite sterile, as did also a specimen of his blood which was incubated for three days at a temperature of 37° C. There are, however, very definite blood changes, and on three separate occasions, examination of his blood has shown a very marked increase in the percentage of large mononuclear lymphocytes, suggesting the presence of a protozoa.

We must conclude, then, that infection by an organism is probable, but as yet the organism is not isolated; that the organism is sometimes present in the blood but probably is not always there; that infection of the "exciting" eye may be, and usually is, endogenous, and the sympathising eye is infected by the blood-stream, the organisms being allowed to grow and elaborate in the damaged uveal tissue of the "exciting" eye, and subsequently conveyed by the blood to the "sympathising" eye.

Treatment.—A well-known ophthalmic surgeon in London once told me that he never allowed a case of sympathetic ophthalmia to occur in his clinic, and in answer to my query as to how he had avoided it, he said that he always removed every eye which had a perforating wound. The attitude taken up by this surgeon represents very well the hopeless condition of a patient suffering from sympathetic ophthalmia with regard to vision; it is a very terrible disease, and when once established treatment has very little effect on its course. I cannot emphasise too much the absolute necessity for watching with great care *both* eyes of a patient who has had a perforating wound in one; I never allow such a patient out of the ward under five weeks, because you can never be sure that he will continue to attend in the out-patient department, or come sufficiently often for the earliest change to be noticed. If after a period of quiescence and recovery the "exciting" eye becomes painful, more inflamed, irritable, and the vision worse, remove it at once. If the "sympathising" eye begins to water and to show signs of photophobia, weakness, and circumcorneal injection, then also remove the "exciting" eye at once. If, however, the "sympathising" eye already has evidence of inflammatory changes present in it, then it is too late to hope to stop the inflammation by removing the "exciting" eye as in the case before us to-day. When definite inflammation has already set in, the treatment already indicated should be vigorously tried and continued for many months. Mercury has an influence on all inflammatory processes, no matter if they are syphilitic or not, and I have found mercury do more good than any other drug. In sympathetic ophthalmia, quinine, salicylic acid, iron, arsenic, and many other drugs have been tried, but I prefer mercury to all others.

May 9th, 1910.

Camphoric Acid in Treatment of Sweats in Tuberculosis.—E. Levi reports twenty cases to demonstrate the efficacy of camphoric acid in treatment of the sweats in phthisis. He gives 2 gm. (30 grains) in two doses during the day or evening, and found it effectual in many cases in which all other measures had failed to relieve. The sweats are probably due to a number of factors acting on the centres for respiration and transpiration, and the camphoric acid seems to influence them all, while it is well borne and causes no symptoms of intoxication.—*Journ. A. M. A.*, vol. liv, No. 17.

CHRONIC MALIGNANT ENDOCARDITIS SIMULATING SPLENIC ANÆMIA.

Dr. F. Parkes Weber exhibited* the heart from a case of chronic malignant endocarditis simulating splenic anæmia. The patient had been a sailor, æt. 28 years, who had visited South Africa several times, and had often been in the Mediterranean and the surrounding countries. He had contracted malaria when in the Straits Settlements about four years before he died. He had stated that he had had gonorrhœa twice but never syphilis, and that when in harbour he used to drink much spirits and beer. He thought the present illness had commenced about two years previously, when purpuric spots appeared on both legs. From that time he had gradually become weaker and paler, and had noticed an increasing swelling (evidently the spleen) in the left side of his abdomen. There had been occasional oozing of blood from the gums and slight epistaxis. He had been treated at several London hospitals, where apparently the diagnosis of splenic anæmia was made.

Dr. Weber said he was much indebted to Dr. H. G. Turney and Dr. G. G. Butler for the following notes of the case when the patient was at St. Thomas's Hospital, August to October, 1908.

The spleen was then much enlarged, its lower limit reaching a level of half-way between the umbilicus and pubes, the splenic dulness extending upwards to the seventh rib in the axillary line. The liver was definitely enlarged. The apex-beat of the heart was felt in the fourth intercostal space, half an inch external to the left nipple line. The cardiac dulness reached from the apex-beat to the mid-sternal line, and upwards to the third space. There was a fairly well-marked apical systolic murmur conducted into the axilla, and there was likewise a systolic murmur at the base, best marked over the "pulmonary area." Treatment by arsenic was continued throughout his stay at St. Thomas's Hospital.

Blood examinations gave the following results:

	August 14th, 1908.	September 23rd, 1908.	October 8th, 1908.
Red cells (per c.mm. of blood)	5,625,000	3,600,000	3,268,000
Hæmoglobin	50 per cent.	50 per cent.	60 per cent.
Colour index	0·4	0·7	0·9
White cells (per c.mm. of blood)	2840	1740	1360
Differential count of white cells:	Per cent.	Per cent.	Per cent.
Neutrophile polymorpho-nuclears	27	28	26
Small lympho-cytes	58	57·5	41·5
Large lympho-cytes	8·5	9·5	27
Large hyalines	5·5	4·5	5

There was therefore at that time a very decided leucopenia, and this was equally well marked after the patient's admission to the German Hospital on July 1st, 1909.

In the German Hospital the patient was extremely pale, weak, and wasted. Both legs below the knees and the lower inner aspects of the thighs were covered with spots of brown pigment. This remarkable brown pigmentation was evidently the result of repeated spots of hæmorrhage into the skin, for amongst the pigment-spots were some fresh red petechiæ. On the arms and trunks there were likewise a few petechiæ. The ankles were œdematous. There was a tendency to bleed from the gums. The heart was somewhat enlarged, the impulse was forcible, and the apex-beat could be located in the fifth intercostal space, just outside the nipple line; there was a systolic apical murmur. On auscultation over the lungs a few dry bronchitic sounds were heard. The spleen was greatly enlarged and hard; it reached to the umbilicus on the right, and downwards into the inguinal region. The liver was likewise enlarged and hard, its lower edge being felt at the umbilical level. Ophthalmoscopic examination showed a few retinal hæmorrhages (Dr. R. Gruber), and there were some conjunctival petechiæ. The urine contained a little albumen, some granular and epithelial tube-casts, and afterwards blood and blood-casts. There was no evidence of active malaria.

* At a Pathological Meeting of the Medical Society of London.

Blood examinations:

	July 1st and 2nd, 1909.	July 19th, 1909.
Red cells (per c.mm. of blood)	1,700,000	1,700,000
Hæmoglobin	30 per cent.	35 per cent.
Colour index	0·9	1·03
White cells (per c.mm. of blood	1900	1200

Differential count of white cells:

	Per cent.	Per cent.
Neutrophile polymorphonuclears	63·5	58
Lymphocytes	29·5	26
Intermediates	4	8
Large hyalines	2·5	7
Eosinophiles	0	1
Mast cells	0·5	0

These differential counts were made by Dr. A. E. Boycott, who added that no nucleated red cells were found. Amongst the erythrocytes were oval forms. Very little polychromatophilia. The coagulation-time of the blood, as estimated on July 21st, by Dr. Engelsmann, with Sir A. E. Wright's coagulometer, was as much as about thirty minutes.

Splenic anæmia was accepted as a "clinical diagnosis" and the patient was treated with an arsenic preparation. He likewise had orange juice, but on July 19th calcium lactate was prescribed on account of the hæmorrhagic tendency. The pulse was usually 100 to 110 per minute. Systolic brachial blood-pressure (July 26th) 105 mm. Hg. During the first ten days in the hospital his temperature was often raised (up to about 101°F.), but after July 10th there was seldom any fever. On the day before the patient's death, which occurred on July 29th, there was, however, again slight fever. Towards the end there was considerable œdema with bronchitic signs.

Necropsy (*about half a day after death*).—The *brain* (weight 51 oz.) showed nothing special, except the small cystic remnant of an old hæmorrhage above the front part of the internal capsule on the left side.

The *heart* was large, weighing 21 oz., and the left ventricle was hypertrophied. The cardiac musculature was very pale. There was old thickening of the mitral valve, and adherent to one part of the valve was a soft organised thrombus of about the size of a hazel-nut. In the left auricle, and apparently growing from the intra-auricular septum was a large polypoid excrescence, which measured about 70 mm. in length and about 35 mm. in breadth. It was attached by a short circular pedicle about 7 mm. in diameter to a spot in the interauricular septum, about halfway between the thrombus on the mitral valve and the (closed) foramen ovale. The free end of this polypoid excrescence, which was evidently an old organised thrombus, was opaque, white and hard, its surface feeling as if it were partially calcified. The lungs showed some recent bright red pleural petechiæ at their bases. There was a moderate amount of serous effusion in the left pleura. There was likewise some ascites.

The spleen was very large, and, together with a small splenculus, weighed 110 oz. There were several subcapsular, non-suppurative, anæmic infarcts, some of which were connected by perisplenitic adhesions to the surrounding parts. On section the substance of the spleen (and also that of the splenculum) had a milky-red appearance, and was rather friable.

The liver was large and hard, weighing 156 oz. The capsule was roughened at parts, and there were some peri-hepatitic adhesions. There were many bright-red recent petechiæ in the capsule. On section the substance of the liver suggested fatty infiltration and chronic passive congestion, together with some increase of fibrous tissue. The gall-bladder and bile-ducts showed nothing special.

The kidneys (weight together 17 oz.) were large and rather pale; the cortical substance was not diminished, and the capsules stripped readily. Petechiæ were noted in both renal pelves. In the right kidney there was a non-suppurative embolic infarction.

There were petechiæ in the mucous membrane of the stomach, ileum, and cæcum, confluent in part of the ileum and cæcum. Some of the lymphatic glands near the hilum of the spleen were enlarged. The splenic and portal veins were very large, but otherwise nothing abnormal was found in the abdominal veins or arteries. The bone-marrow of the shaft of the left humerus was examined and found to be quite red.

A microscopic examination of pieces of the spleen, liver, kidney, and bone-marrow (from the shaft of the humerus) was kindly made by Dr. J. C. G.

Ledingham. All four organs presented as their most conspicuous feature a plasma-cell development with the elaboration of new fibroblastic tissue.

Dr. PARKES WEBER thought the decided leucopenia and chronic splenomegaly in this case seemed to point to a diagnosis of splenica anæmia or Banti's disease, but the post-mortem examination showed that the fatal illness was really a chronic form of malignant endocarditis, such as has recently been so well described by Osler ('Quarterly Journ. Med.,' Oxford, 1909, vol. ii, p. 219) and Horder (*ibid.*, vol. ii, p. 289), which amply accounted for the chronic purpura and remarkable cutaneous pigmentation of the legs. It has, indeed, been abundantly proved that leucopenia is not pathognomonic of splenic anæmia (if splenic anæmia can be supposed to exist at all as a distinct disease). It is, indeed, doubtful whether splenic anæmia (of adults) should be regarded as anything more than a group of symptoms, which may be set up by various pathogenic agencies. Not only are there cases of congenital or acquired chronic acholuric jaundice with splenomegaly and anæmia, which might be wrongly regarded, owing to the temporary absence of obvious jaundice, as examples of "splenic anæmia," but in many cases of supposed splenic anæmia and Banti's disease a necropsy has given evidence of considerable vascular or endocardial disease, probably of infectious origin. In one of the earlier fully described cases in England, that of Dr. S. West ('Med.-Chir. Trans.,' London, 1896, vol. lxxix, p. 323), there was evidence of considerable old disease of the aortic valves of the heart, and the patient had a septic pyrexia at the end. In several published cases of splenic anæmia thrombotic obstruction of the splenic and portal veins or disease of the splenic artery has been found at the necropsy.

Dr. DE HAVILLAND HALL agreed with Dr. Parkes Weber in regard to the difficulty of diagnosis of splenic anæmia, and he somewhat shared the view that the disease, splenic anæmia, was one which was not based at present on any very sure foundation. Certainly the case which he had brought forward that evening would warrant some scepticism in the future in doubtful cases of so-called splenic anæmia, and would lead one to be extremely careful in investigating the condition of the heart and considering the possibility of it being due to ulcerative endocarditis rather than to an infection of the spleen. He was particularly interested in that question because he had been seeing in consultation a lady in whom he diagnosed splenic anæmia when he first saw her. But on his last visit, a month or six weeks ago, he felt inclined to modify his diagnosis, inasmuch as the spleen became very irregular, painful, and was evidently adherent to the abdominal parietes, suggesting the possibility that it was malignant disease of the spleen. As he had not yet seen malignant disease of the spleen he hesitated to make the diagnosis. He scarcely thought his case was one of ulcerative endocarditis, though the patient had a murmur at the apex. There were some cases which seemed to correspond to the clinical description of splenic anæmia which had been operated upon. Eighteen months ago he had a case in Westminster Hospital in which Mr. Spencer removed the spleen, and the patient made a most excellent recovery, and when seen six months after the operation she was in very good health.

Dr. PORTER PARKINSON asked whether Dr. Parkes Weber considered that leucopenia was moderately common in malignant endocarditis. His attention was drawn to the possibility of it by having a case of the kind himself which died three or four weeks ago, and in which during life there was a constant leucopenia, though not to the same degree as in Dr. Parkes Weber's case; not only was the leucopenia present during the last week or two of life, but also during the previous three months. The post-mortem examination showed malignant endocarditis. The symptoms during life were very few. The patient had constant fever, enlargement of spleen, and purpuric spots in various parts of the body. He suspected, owing to the presence of heart disease, etc., the possibility of malignant endocarditis for a long time, but he was doubtful for a time because of the leucopenia which existed. He expected to find leucocytosis. Cultivations from the blood showed the presence of quantities of small streptococci, and so the diagnosis was made during life in that way, and was confirmed afterwards. It seemed most unusual, and he had not met with it before in a septic illness of that kind.

Sir JOHN BROADBENT said he had a very interesting letter last year from a doctor in Toronto referring to a case of a boy, æt. 14 years, which

was in St. Mary's Hospital fourteen years ago, and which was then diagnosed as splenic anæmia. The characteristics of the case at that time were enlarged spleen, with melæna and extreme anæmia. The interesting point was that this patient was alive fourteen or fifteen years afterwards. He was in a hospital at Toronto with jaundice and extreme anæmia. Usually such cases were regarded as going on fairly soon to a fatal issue. Apparently this patient remained well for fourteen years, and then had a relapse with the same kind of symptoms at the age of twenty-eight.

Dr. SAMUEL WEST said that the question of splenic anæmia was a very interesting one, and well worthy of further discussion. But there were difficulties in deciding what was meant by splenic anæmia. Probably most people would agree that if the definition were strict the disease was quite rare. With regard to its relation to malignant endocarditis, there was another difficulty, namely, as to the sequence of events, *i.e.* as to which disease preceded, because it was possible that the infection which led to malignant endocarditis might occur as a secondary complication. A difficulty which he did not see his way to overcome was the curious condition of the spleen, for in malignant endocarditis there were often definite infarcts in many parts of the body as well as in the spleen, yet in typical splenomegalie primitive, infarcts in the enlarged spleen or other parts of the body were very rare.

Dr. PARKES WEBER, in reply to Dr. Porter Parkinson, said he did not know in what proportion of cases of malignant endocarditis leucopenia was found. Leucopenia not rarely occurred in cases of chronic enlargement of the spleen due to malaria or other causes. It was likewise a recognised feature in the course of certain acute infections. The question which Dr. West mentioned, about the relation of splenic enlargement to malignant endocarditis was one which could not be discussed quickly. No doubt there were cases of chronic malignant endocarditis in which the malignant endocarditis was secondary to some infective focus elsewhere in the body. He certainly did not wish to suggest that the majority of cases which were clinically diagnosed as splenic anæmia were cases of malignant endocarditis. He believed, on the contrary, that cases due to malignant endocarditis only formed a small proportion of the cases which were clinically diagnosed as splenic anæmia, and the case mentioned by Sir John Broadbent was probably not one of malignant endocarditis.

HYPERNEPHROMA: TUMOURS OF BOTH OPTIC NERVES AND HYDROCEPHALUS, ASSOCIATED WITH OBESITY AND SIGNS OF PRECOCIOUS DEVELOPMENT.

DR. LEONARD GUTHRIE and Dr. W. D'ESTE EMERY exhibited* specimens from a case of hypernephroma, tumours of both optic nerves and hydrocephalus, associated with obesity and signs of precocious development.

Dr. GUTHRIE said the child was a girl who was admitted to the hospital in October last on account of chronic hydrocephalus. She was four years of age. She was a full-term child and the labour was normal. The head was large at birth, but the child had always been considered intelligent, although she did not walk until she was eighteen months old. Her sight had always appeared deficient, but since April last she had become practically blind. Her health, as a rule, had been good, except that she suffered frequently from attacks of headache and vomiting. On admission she was found to be very stout and heavily built, a big child for her age. The head was very large and the frontal and occipital regions were bulging. The head was long rather than broad. The circumference was 21¾ inches and the measurement from ear to ear 12½ inches. With regard to her mental condition, she was bright and talkative and intelligent, but her speech was indistinct and somewhat idioglossic. The pupils reacted normally; there was a regular slow nystagmus, with rolling of the eyeballs in the way peculiar to sightless children. There was a varying nystagmus, but no paralysis of any of the ocular nerves. She was almost blind, but could distinguish light from darkness, between an electric lamp which was alight and one which was not lighted. There was advanced primary optic atrophy of both optic nerves, but no paralysis of

* At a Pathological Meeting of the Medical Society of London.

any other cranial nerves. No tremors and no weakness of the limbs. She could sit, stand, and walk with guidance. Her gait was staggering, but not inconsistent with the blindness. The tendon reflexes were exaggerated in the legs. There was no ankle clonus. The plantar response was flexor, heart and lungs normal, pulse 112, respirations 36, urine normal. She was in good health at the time of admission. By the end of the month her condition was worse; she was no longer bright and talkative but was drowsy and lethargic, and would not sit or stand or talk of her own accord. Headache was constant and vomiting occasional, but neither of them very severe. By degrees she became unable to stand or walk, and after a time any attempt to make her do so caused so much distress that the attempt had to be abandoned. Her symptoms were increasing intra-cranial pressure, and therefore lumbar puncture was done on two occasions. At the first puncture clear fluid was withdrawn, which was found to be sterile and there were no cells. As there was no relief a week later lumbar puncture was again done, but this time no fluid could be withdrawn. She became more drowsy and lethargic than before, and the headaches and vomiting continued. It was therefore resolved to puncture the lateral ventricle, and it was done by Mr. John Murray six weeks after her entry into the hospital. About two ounces of fluid spurted out, after which the brain sank and pulsated freely. The child never recovered consciousness. In the evening she was convulsed and rigid in the upper extremities, and there were jerking movements in the lower limbs. She lingered on for another twenty-four hours and then died, with a temperature of 107° F. At the post-mortem examination her general appearance was that of a short, stout girl on the eve of puberty, rather than that of a child æt. nearly 4 years. There was a constriction at the waist; the pelvis was larger and the hips wider than normal for her age. The circumference of the pelvis between the anterior superior spines and the great trochanter was twenty-two inches. The vulva, the labia majora and labia minora and the hymen were well developed. The contour of the limbs approached the adult type, the thighs were large, and the femora sloped inwards from the hip to the knee more sharply than normal for her age. The breasts were not enlarged and there was no hirsutes, either general or localised. There was advanced internal hydrocephalus, and two symmetrical tumours of the size of small marbles, each occupying one optic nerve, and a hypernephroma of the size of a large grape, occupying the site of the left adrenal capsule, were found. The left ovary was large, but on examination it showed no signs of ovulation. The signs of precocious development in this case were slight, and they would not have been deemed worthy of record if they had been noticed only at the post-mortem. The case brought forward additional evidence of the fact now recognised that hypernephroma or tumours of the adrenal cortex were associated with precocious sexual and physical development.

There were two varieties of precocious development observed in cases of hypernephroma. One of them was termed "herculean" and the other the "precociously obese type." The former involved the muscular and osseous tissue, and was associated with extreme muscular development and great development of the sexual organs. That was a more common type in boys than in girls, but it also had its counterpart in girls, who to all appearances look like minature women.

A picture of a case shown by Mr. Hugh Lett before the Society for the Study of Disease in Children was exhibited, and another of the obese type similar to the case under consideration. The child was æt. 4¾ years, and looked more like a brewer's drayman than an ordinary boy. There were masses of fat between the shoulders, but the sexual organs were infantile. There were some signs of hirsutes about the pubes. A slide of a girl, æt. 3 years, was also shown. She looked like a woman of middle age, and had striæ about the thighs and abdomen. The cheeks were swollen with fatness and were dusky red in colour, and studded with stellate veins. The hirsutes with extreme muscularity or obesity was suggestive of hypernephroma. A table giving particulars of twelve other cases was also shown. The most important of them was a child æt. 4 years, who had marked obesity and hirsutes, with the adult type of genitals. She menstruated at the age of three and there was the adult type of mammary enlargement. In one case there was no menstruation, but the child suffered from retention of menses, and after death a hæmatocolpus was found. Apparently there was complete sexual maturity in only three cases. The

last case on record was that of adrenal ovarian rest. That subject suffered from metrorrhagia and menorrhagia at the age of three years and nine months.

Other tumours of the ovary regarded as sarcomatous or carcinomatous and associated with precocious development might possibly be of the nature of adrenal rests, but certain ovarian tumours, such as cystic dermoids, usually led to sexual infantilism and retardation of growth rather than to the opposite.

Precocious development and obesity with or without precocious sexual development should suggest hypernephroma. With regard to the frequency of the association, in 35 cases in males there were only 4 in whom the presence of hypernephroma was proved, 3 of the herculean type, 1 of the obese. In 100 females who were subjects of precocious sexual development only 12 suffered from hypernephroma. But in the vast majority of cases of precocious sexual development the history was absolutely unknown. The post-mortem records were very scanty and the history only amounted to isolated observations during life. Probably the majority of the cases grew up, and after they had reached the age of puberty they were no longer wonders and led a normal and healthy life. It was scarcely conceivable that those cases all suffered from hypernephroma, as otherwise they would all die. But as it was known that precocious development and physical development were associated with hypernephroma, it was conceivable that some abnormal condition of the adrenal cortex might be responsible for all cases of precocious sexual and physical development. The point of interest in the present case was the hydrocephalus, the tumours of the optic nerves, the obesity, and the physical growth with the slight precocity of the sexual organs. With regard to the relations of hydrocephalus to precocious development, Dr. Guthrie had collected three cases in which the association was found. They might have been coincident. With regard to the relation of optic nerve tumours to hypernephroma, Dr. Guthrie said he would leave Dr. Emery to give his views on that matter.

Dr. EMERY, in demonstrating the specimens, said that the adrenal tumour was almost spherical being about one and a half inches in diameter, and that it grew from the left suprarenal capsule at its lower and inner extremity. At this point the suprarenal substance had disappeared, and this seemed to have been brought about by pressure rather than by infiltration. The specimen looked as if the tumour had developed outside the adrenal and had pressed against it and destroyed it. That this was not the case seemed proved by the fact that the tumour had a definite capsule, in which definite suprarenal elements (cortex) could be seen under the microscope. The structure of the adrenal tumour was that of a fibroma or fibro-sarcoma. In some places it consisted of well-formed fibrous tissue of scar-like consistency, whereas elsewhere it was softer and the cells more spindle-shaped. Besides this there were remarkable giant-cells, some of which were as much as 100 μ in diameter. They had one, or more usually, several nuclei, but these were never arranged in the manner seen in tuberculous giant-cells.

As regards the brain tumour Dr. Emery could not say as much about it as he would like since its macroscopic appearance was almost exactly that of the surrounding brain-tissue, and he was therefore unable to give its exact limits until the whole organ had been cut to pieces. It was quite symmetrical and occupied the region of the optic chiasma, which it apparently destroyed and replaced. It extended forwards along the optic nerves as far as the optic foramina, where it stopped abruptly. It extended backward into the crura and outwards into the region of the fissure of Sylvius, but its outer and superior limit could not be defined with exactness. It was slightly firmer than normal brain substance, and this was the only criterion in which it could be defined therefrom. Its structure appeared to be that of a glioma, but on careful search a very small number of giant-cells similar to those seen in the adrenal tumour were found, suggesting that the two were in some way related, though which was the primary and which the secondary was difficult to say.

There was little to be noted in the other organs. The ovaries appeared large for a child of that age, but were normal in structure; the vagina and uterus were normal, as were also the pineal and pituitary bodies, the thymus, thyroid, etc.

Dr. PARKES WEBER said he could not help suggesting, with regard to the sections which had been so well demonstrated by the epidiascope method, that the peculiar large cells in the tumour-

tissue represented the chromaffinic cells of the adrenal medulla; perhaps there was something in their deep staining which gave rise to that suggestion. He asked Dr. Guthrie whether there were any signs of excessive adrenalin action in the patient during life; for instance, was there unusually high blood-pressure?

Dr. LAKIN said that in children who had had nephroma there had been found to be multiple deposits in the bone. He asked whether that was also so in adults.

Dr. GUTHRIE, in reply, said he could not say that any scientific investigations of the blood-pressure were made, but the tension of the pulse was not high. He believed that as a rule in such cases it was low. In such cases he had no information that deposits ever occurred in bone, but it seemed natural that it might be so, because there might be metastasis from the tumours to different parts.*

Dr. EMERY replied that he had thought of the question of there being chromaffinic cells, but there was no evidence for or against this view. As to whether there would be adrenalin poisoning if they were so, it would depend on whether they discharged their function. As a rule, he thought tumour-cells did not discharge their function (*e.g.* an extract from adrenal tumours had no effect in raising blood-pressure), and he did not think the suggestion gave any help one way or the other.

Dr. WEBER reminded Dr. Emery that occasionally tumour-cells did, to some extent, retain the functional activity of the cells of the organ from which they were derived; for instance, in certain primary carcinomata derived from the glandular cells of the liver some of the tumour-cells were still able to excrete bile.

BRONCHIOLECTASIS.

DR. W. D'ESTE EMERY and Dr. F. S. LANGMEAD described† a case of bronchiolectasis.

Dr. LANGMEAD said the child died six days after the illness started. The patient was a boy, æt. 4 years, who came to Paddington Green Children's Hospital very short of breath, with the accessory muscles of respiration actively working and blue. The history which was given by the mother was that the condition had come on somewhat acutely three days before. On examining the child he could find no evidence of pneumonia, and the symptoms were not those of that disease. Although there was urgent dyspnœa the condition was not painful, and there was no expiratory grunt; there was no flushing of the face, but simply a dusky condition. On examination of the lungs only a few crepitations were found at the base. Seeing that there was so much cyanosis and the child was very ill, he provisionally diagnosed acute generalised tuberculosis of the pulmonary type, and the child was admitted into the hospital. After having stayed there for a day the cyanosis was very marked and the asphyxia was more striking, and the child died three days later with all the evidence of extreme respiratory distress.

Dr. EMERY demonstrated the lungs, which showed a series of what appeared to be cystic spaces closely crowded together throughout both lungs. He said he ought really to apologise for the name bronchiolectasis; and wished to substitute for it a name which did not imply a definite diagnosis, namely, "honeycomb lung of acute formation." Under "acute bronchiolectasis" he thought several different diseases had been described. In one case which was described before the Pathological Society about two years ago by Dr. Bernstein there was apparently a similar lung to that in this case. That case was also a child, and the condition had been rapidly fatal. In that case Dr. Bernstein was able to demonstrate that there was emphysema as well as a certain amount of bronchiolectasis. But in other cases of bronchiolectasis which had been described no structure which corresponded to the bronchus had been demonstrated, therefore it was questionable whether they should be properly so-called. The commonest cases where that occurred were those of acute generalised tuberculosis. Other cases in which the condition was found were multiple abscesses, in which the contents had been absorbed, and such a case was one described before the Pathological Society by Dr. Tooth, and that was the declaration of a Committee which reported on the case. It was a particularly uncommon condition. Other

* *Note.*—Dr. Robert Hutchison has recorded cases of supra-renal sarcoma with metastasis in the skull in children, but in none was there any indication of premature sexual development ('Quart. Journ. of Med.,' vol. i, No. 1, October, 1897).

† Delivered before the Medical Society of London.

cases, such as the one shown that evening, were those which were simply acute emphysema, in which no structure which could be said to be bronchial could be demonstrated. Under the term "honeycomb lung" he thought there were two varieties, acute and chronic. Dealing with the acute, he thought there were several different histological changes. There was the acute bronchiolectasis properly so-called, there was acute emphysema, there were cases of acute miliary abscesses as a part of broncho-pneumonia, and also cases of acute miliary tuberculosis, and those were the commonest of all. Dr. Emery also demonstrated an epidiascope representation of one of the cysts. The cysts varied in size. The largest was a quarter of an inch in diameter. In some of the cases which had been described there had been larger cysts and fewer of them, but most of the cases of which he had found records had lived longer than in the present case. When the specimen was quite fresh it was a very striking one, the whole lung being studded with those cavities. But they did not occur in the ordinary positions for emphysema. There was less at the anterior margin than at the apex. The place where the condition was most abundant was on the diaphragmatic surface of the lung, and in the sulci between the lobes. The slide showed the general appearance of the lung. The cysts were quite round before the lung was hardened. The alveoli in the neighbourhood of the cysts were not much altered in size, but their walls were thin, in some places extremely so, and this was especially marked in close proximity to the cysts themselves. There was definite proof that the cysts were not due entirely to the dilatation of any pre-existing structures, whether bronchioles or alveoli, in that projecting into some of them were thin membranes, evidently all that remained of the alveolar wall. He thought the condition might possibly be a sequel of a pneumonic process. It was known that the removal of the exudate in the alveoli of the lung after pneumonia was due to the action of a proteolytic enzyme, and it seemed possible that in this case this ferment had not been content with doing so, but had gone on to digest and destroy the alveolar walls themselves. There appeared to be some thrombosis of the vessels, and this might account for the process, the vitality of the tissues being impaired owing to the loss of blood-supply. The dilatation of the cavities thus formed he thought to be secondary, and compensatory to the collapse of other areas of the lung.

May 9th, 1910.

COAL-TAR DERIVATIVES VERSUS OPIATES.

SAJOUS points out that the history of coal-tar derivatives as therapeutic agents shows that it is not through the medical profession that the sins attributed to them have been perpetuated, but through lay manufacturers of headache powders. If such a reason were to prevail among those who oppose the use of acetanilid, antipyrin and acetphenetidin, in practice, or their retention in the Pharmacopœia, then all our valuable therapeutic agents should be similarly treated, since all in turn have once on a time been used by empirics or enterprising commercialists who look on human suffering merely as an opportunity to satisfy their greed. Even the fate of opium and its analgesic salts would be sealed by the evil reputation soothing syrups have given them.

The most important phase of the question which bespeaks their retention among official remedies, however, according to Sajous, is the relatively small proportion of coal-tar habitués as compared to the victims of the morphine habit. Even the most active agent of this class used in medicine, acetanilid, is practically inoffensive as a cause of drug mania. Withdrawal of coal-tar products gives very little trouble. Under the gradual withdrawal of the drug, tonics, and the bromides, and with a little cannabis indica to subdue pain, if any, prompt recovery ensues.

Much is said of the poisonous effects of coal-tar products. If, however, we study closely the causes of the deaths they have produced, we shall find that in practically every instance the fatal results were due to their use either in excessive doses or in disorders in which experience has shown them to be contra-indicated. The fatal results due to excessive dosage, or to the cumulative action of too many doses within a given time, belong to the category of self-treatment by laymen and the free dispensing of proprietary drugs—features which stand outside the pale of professional responsibility, though well within the reach of legislation.—*Journ. A. M. A.*, vol. liv, No. 17.

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CONTENTS.

PAGE

***A Clinical Lecture on Some Cases of Tubercular Disease.** By F. C. WALLIS, F.R.C.S. 81

***Asthma.** By H. BATTY SHAW, M.D., F.R.C.P. 86

***Pituitary Extract and some of its Effects.** By LEONARD WILLIAMS, M.D., M.R.C.P. 92

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A CLINICAL LECTURE

ON

SOME CASES OF TUBERCULAR DISEASE.

Delivered at Charing Cross Hospital.

By F. C. WALLIS, F.R.C.S.,
Surgeon to the Hospital.

GENTLEMEN,—This woman, whom you have seen before, has tuberculous disease of the knee-joint. There are three cases of tubercle I want to show you to-day. This particular knee-joint, although not the best example, is an instance of erasion of the knee-joint. As you will see, she has quite a good joint, and she can bend her knee through a certain angle. I have not done what may be termed a classical excision of the knee-joint for a large number of years. I show you the incision by means of which the joint is opened. The incision is taken straight over the patella, the patella is sawn across, and the incision is continued to the aponeurotic extensions of the quadriceps extensor on either side, so that the joint is freely opened from the front. The knee-joint is then flexed. You can in that way see the whole of the articular surface of the bones forming the knee-joint. You can also see the extent of the disease of the cartilages and synovial membrane. The former notes of the case, I am sorry to say, have been lost. It is two years ago since the patient left the Hospital after operation. This patient has not any disease of the articular cartilages, and she has very fair movement in her knee-joint. If she had erosion of cartilages she would have now either fibrous or bony union of the approximated bone surfaces. In doing this particular operation, you remove all the diseased portion of the synovial membrane, and the cartilage where it is involved; the tubercular

deposit in the bone when present may descend into the tibia, or ascend into one or both of the condyles of the femur for some distance. In such cases a thorough erasion and eradication of the diseased portions which are seen are made with scissors and knife, spoon, etc., and finally the whole surface is swabbed over with pure carbolic, and the joint sewn up. After that the patella is wired and the skin edges brought together, a drain is left in for twenty-four hours on either side of the transverse incision. This is very necessary, because the exudation after that violent irritant carbolic acid is profuse. It drains very freely, and the hyperæmia producing this serous flow is one of the elements of success in the results. I have done this operation for knee-joint diseases now for fourteen years and in quite a number of cases. The only bad results I have had are this one, which you see now, and one other, which I think is as bad as this one. I have seen my cases from time to time, and they may get some thickening, possibly some recrudescence of tubercle which has been left. But, except for these instances I have not had any failures. Cases may return with perhaps a little pain or swelling, but after rest and appropriate treatment and applications the swelling has subsided and one has not heard any more of it. This patient has a sinus, and she has had it much as it is now for a year. Her doctor sent her up to me to see what I could do for her. It must be something particularly localised or it would have been worse than this after a year. The track goes down, not to bone, but to some portion of tuberculous material which was probably left over from the previous operation. I am trying her with what is known as Bier's treatment, which we have used with great success in other similar cases. My reason for taking up this Bier treatment is that Dr. Poyser, my house-surgeon twelve months ago, was very keen about it, and did good work with it whilst he was in my wards, and he wrote a very excellent thesis on it, which I read with great interest, and which you may be able to read later on in one of the journals. I recommend Bier's method of treatment to your consideration for certain instances of chronic inflammatory trouble, such as stiff (not ankylosed) joints, and, in fact, all those cases which are benefited by an increased blood-flow for the time being, that is to say in a condition of recent fibrosis, particularly in the joints and muscles, or in tendons around joints. These are very much benefited by the Bier treatment. Boils and carbuncles are also extraordinarily well treated by it. Mastitis it is also good for, and one might enumerate most subacute or chronic inflammatory states in which this congestive treatment is useful.

Case 2.—The next case is also a tuberculous patient, and she has had a very much more trying condition, that is to say, a tuberculous wrist-joint, a very trying and troublesome complaint. This is the second time she has been in the hospital. The first thing she noticed was a small sore on the under part of the wrist, below the thumb. When she came into the hospital in June this year she had a most appalling condition of advanced tuberculosis of the wrist-joint, the most advanced I have ever seen. There was a sinus which went down to the bone, a large puffy swelling, the skin was blue and livid, and there was this lesion which you see now. She looked very ill indeed, and thin, and the joint was causing her extreme pain, which deprived her of sleep at night. Altogether it was a source of very great distress to her. I made this longitudinal incision over the wrist, which is more or less the incision of election in ordinary excision of wrist-joint. You know the wrist-joint consists of a number of small bones, and there are numerous synovial membranes which can become infected gradually in tuberculosis of this joint, and the disease generally starts in one or other of the synovial membranes of the wrist-joint. Gradually the bones and joints are involved, and small chronic abscesses form; these unite, and the tuberculous material ramifies all over the place in such an advanced case as this. When it is incised a pulpy mass of degenerated synovial membrane is seen and carious and necrosed bones. With this longitudinal incision which I have mentioned you do not divide any tendons, but you can expose the whole of the wrist-joint and remove the infected tissues. The difficulty in such an advanced case is to get rid entirely of the tuberculous disease. And in this instance it was extremely difficult, because not only was the whole joint gone, as far as the bones are concerned, but there were two sinuses as well. We operated upon it and swabbed it out with pure carbolic, and left a drain in the posterior part, and the wound was allowed to granulate up. It is now four or five months since

she left us, and she has come back because of a small sinus. There is very little power in the hand so far. There is but little power of extension of the wrist. As far as general healing goes it is quite a good result. The time is hardly ripe to say anything about the result of passive movement and massage, which are so useful in these cases. She also is having Bier's treatment for producing congestion of the parts, and certainly it has done her good. Another way of treating these stiff joints is to put them into a hot-air bath. When they come out of that they are very pliable, and fairly free movement can be started with them. The stiffness returns to a certain extent when the joint cools down again. But by taking them each time a step further in movement progress is made. If she can get some power in the wrist and is able to do needlework and approximate the thumb and fingers, she will have a very good result having regard to the severity of the condition when she first came to us. Another point with regard to this patient is the fact which you must always have in mind with regard to tuberculous joints; namely, that the most important asset with regard to the recovery of these people is their general health. Improve their general health by good food and tonics and fresh air. Warmth is also good for them, and when they start getting better the whole thing goes together, the joint improves, and the patient improves, and even if there is some set-back, as this woman has had, the improvement in general health enables her to tide over it. I have no doubt she will get well, and we shall be able to instruct her how to gradually increase the power of her fingers. I hope she will come and see us again later on and show us how far the movements have progressed.

Case 3.—This girl came in originally with tuberculous disease of the hip-joint on the left side. When she arrived, her hip-joint was flexed at an *acute* angle with her pelvis and it was adducted and inverted. When she went out she had a perfectly straight leg and could lie flat on her back, and we were all pleased with the result. She was put on a Thomas's splint. But, as is typical in these cases, the first thing done was to throw away or pawn the splint, and so gradually she relapsed, and has now returned in exactly the same state for which she came in some months ago. You see here rather a typical condition of hip-joint disease. Notice the atrophy of the muscles of the left leg. The left anterior superior spine is tilted up to a very marked extent, some two and a half inches, and her back is very much arched as she lies at present. There are two schools of treatment for this form of hip-joint disease; there is the operative method, and the expectant method. If you want to see the results of the expectant method, go to the Alexandra Hip Hospital in Queen Square. There they practically never do excision of the hip-joint, they put the children on to the best diet they can get, put them on extensions, give them rest, good food, etc., and if an abscess forms in connection with the joint it is opened and injected with iodoform emulsion, or scraped and injected, and closed, and if another abscess forms it is treated in the same way. This treatment has been in existence for some twenty years, and the results have been published, and they are excellent. I do not know whether any of you have seen the after-results of excision of the hip-joints. It is a mutilating operation, and even at that the results are not always satisfactory. It is difficult to get all the disease away, and if this happens to be in the acetabulum as well as in the head of the femur, it is *extremely* difficult to get the whole of the disease away. This child is æt. 6 years, and if I were to remove the head of the bone and scrape her acetabulum, what would be the result? She would have a distressingly flail-like limb for the rest of her life, and as the other limb grows naturally there would be a greater and greater difference between the two limbs, one of which would be practically useless. If I can get the limb into good position again and fix it, and if she be taken from her surroundings for two or three years, this child will grow up a fairly well-built girl, healthy and useful for the purpose of getting her livelihood.

Sir William Treloar has recently started a home for crippled children down at Alton, a charming place, with a hospital for such patients as this child. There is a convalescent department, with schools, workshop, play-fields, and everything that a child could want, including the opportunity of learning a trade. I propose to take advantage of the fact that I am on the staff of that hospital to get this little child there in an unmutilated condition. It will be a boon, and a very interesting example of what you can do with a child like that

under the conditions which Sir William Treloar has provided. Compare that with what this child would have to go through if she is left alone as she is now, or even if I put her right again and send her back to her people!

Case 4.—The next case I wish to talk about is an interesting one. A man was brought in acutely ill and suffering from an ischio-rectal abscess. The ischio-rectal abscess was seen by the house-surgeon who very properly and promptly operated upon it. The patient died in spite of this a few hours after admission. There has been a post-mortem examination as you see, which shows a cirrhotic liver and

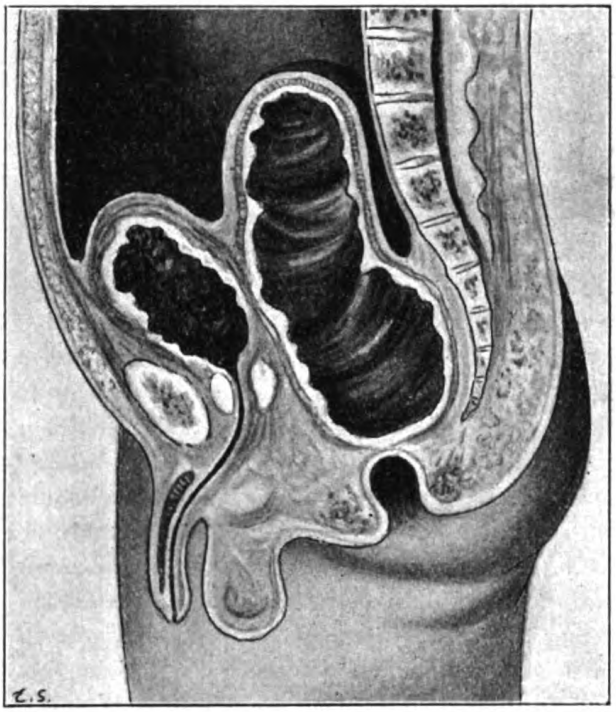

Fig. 1.—Showing the proctodæum and blind end of gut.

granular kidneys, and the pelvis of one kidney is dilated. The track of the abscess appears to go into the prostatic urethra. This is unusual. I want to tell you something about these abscesses because you are sure to have to deal with them in practice, and sometimes, as in this instance, the results can be most serious.

During development the lower opening of the bowel is formed by the fusion of the proctodæum with the blind end of the gut (Fig. 1).

In Fig. 2 you will see just where the rectum and anus meet. Here are various folds, called crypts of Morgagni, and below is the anal mucous membrane. You can understand how this puckering arises. If you have the large blind end of the gut coming down to meet this small proctodæum, the blind end of the gut must adjust itself to the small anal proctodæum, and so these crypts of Morgagni are formed. Around this fusion line are the sphincters in a state of chronic contraction. When the bowels act the contents of the intestine pass over this area and the sphincters are dilated. If some very constipated action or foreign body passes over here, it often gets on to one of these crypts, and tears it down. There may be a little blood passed, and some sensation of discomfort, but not necessarily anything more. Suppose an individual is chronically constipated and has a large amount of fæcal matter in his large bowel, which is loaded with micro-organisms, or his opsonic index is too low, or he has had for many

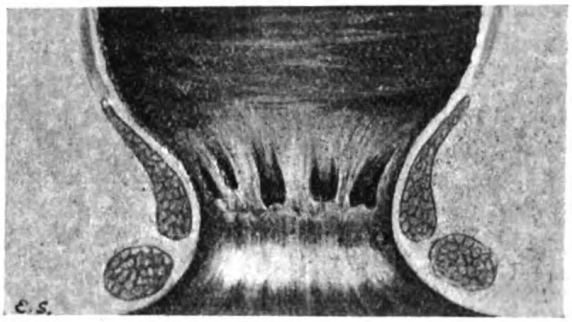

Fig. 2.—Showing crypts and columns of Morgagni.

years a chronic condition of pyorrhœa alveolaris, and has streptococci in his fæces as well as in his blood. The result is that if there is a torn-down pocket here, a sort of test-tube is formed in which these organisms can multiply as they did in this case. They may go up underneath the mucous membrane and form a submucous abscess, or travel out between the sphincters, and into the ischio-rectal fossa, and form an abscess, varying in size, which would be either opened or burst externally, leaving a track leading from outside to inside. Or it may push up the mucous membrane and form a submucous abscess lying between the mucous membrane and the internal sphincter. Or it may go up and form a pelvi-rectal abscess running between the internal sphincter and the levator ani into the pelvi-rectal space, and here there is nothing to prevent enormous abscesses forming. This is a serious form of abscess. At

the back, between the sacrum and the bowel, there is a lot of loose areolar tissue, and an abscess may form here also. Abscesses may occur in five different situations, viz. (1) sub-tegumentary, (2) ischio-rectal, (3) submucous, (4) pelvi-rectal, (5) retro-rectal (see Fig. 3). Remember that these injured crypts of Morgagni are suitable breeding-grounds for micro-organisms; the pus once formed tracks into one of these places. I have not time to go into all the signs of them to-day, but remember in connection with them the fact which I first pointed out many years ago, and which is now accepted by most, viz. that the initial lesion

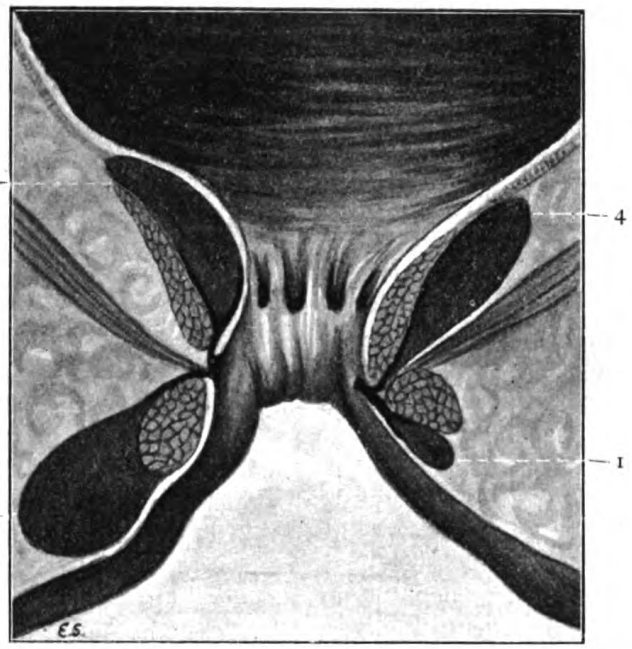

FIG. 3.—1, Sub-tegumentary; 2, ischio-rectal; 3, submucous; 4, pelvi-rectal.

in all these cases is at the ano-proctodæal junction. The point in regard to diagnosis and treatment is this: that when you have a patient coming in with ischio-rectal abscess, or if he comes to you with the history that he has a good deal of pain, and feeling of weight in the bowel, and that when his bowel acts there is a good deal of pain, which lasts afterwards, and he may be passing some discharge, either purulent or bloody, and the temperature may be raised, *never* omit to examine him. There will be perhaps submucous swelling, just obvious, and if you compare one side with the other, you will find a difference between them. If it is an ischio-rectal abscess it will point well out, and an inspection of the buttocks will show you a difference on the two sides, and the patient will tell you one side is painful but the other is not. The sub-tegumentary form is quite easy to recognise. The pelvi- and retro-rectal varieties are the most difficult to diagnose. If you have a patient with pelvi-rectal abscess you will probably find that there is no external bulging, but there is a tender and boggy feeling in the rectum on the affected side as compared with the other. This is a condition it is most important to diagnose. An ischio-rectal abscess bursts externally as a rule. If there is a pelvi-rectal abscess, instead of bursting outside it bursts back high up into the bowel; this causes relief of tension and the patient feels much better. If a doctor examines such a patient and has not the knowledge which you now possess, viz. the fact that all these things originate in these crypts of Morgagni, where the original internal opening exists, and he only finds a large, ragged, obvious opening high up in the bowel, he would say this is the internal opening, and he would start to cure it —as has been done by some—by making an incision which would include all the intervening tissues, and by so doing he would cut through the internal sphincter, the levator ani and the external sphincter, and probably give the man either incontinence for life or greatly impaired controlling power over his bowel. This should never be done,

no matter how long you have to wait for a cure. If you get a high-lying opening from an abscess in connection with the bowel, it is always secondary to the original infection. It is the *original* opening which you will have to deal with, to cure, what is always the result of a large abscess or even a small one, namely, fistula You will have to divide the tissues lying between the *original* infected spot and any opening outside. Do not touch the high-lying opening in these abscesses; this will gradually granulate down and heal on its own. But if, with an excess of zeal and want of knowledge, you cut all these controlling muscles, the patient will regret it, and so will you, because the results of that procedure are most disastrous. These ischio-rectal complaints are conditions which all medical practitioners are called upon to deal with, and you will find that rectal surgery comes largely into your general practice, and if you take every opportunity of learning about it, and becoming thoroughly acquainted with the conditions of their treatment whilst you are in hospital, you will have cause to be thankful, whereas if you do not you will have many bad moments from failing to recognise these points I have been talking about.

May 16th, 1910.

Nephrotomy by the Silver-Wire Method.—The silver-wire method was used by Richardson with results that demonstrate two points: First, the value of the silver-wire method of nephrotomy in certain cases; and second, that the method very clearly has a limited field of application, and should not be employed in every case in which nephrotomy is indicated. Whenever abscesses, fibrous tissues, or other pathologic conditions in the kidney render it necessary to exert strong traction on the wire in order to bisect the organ, many vessels will be lacerated, and consequently both the damage to the kidney parenchyma and the hæmorrhage will be greater than if a knife were employed. The pulmonary complications in the second case following operation show conclusively the dangers of omitting compression of the vessels at the hilum, and thus permitting free renal circulation while doing a nephrotomy on an infected kidney. Richardson feels sure that in this case an infected embolus was swept into the renal vein, thence to the vena cava, right heart, and finally lodged in the right lung, where it produced a localised pneumonia. —*Journ. A. M. A.*, vol. liv, No. 16.

ASTHMA.

A Clinical Lecture delivered at University College Hospital.

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THIS subject has been chosen for you, because it is very probable that many of you have little other knowledge of it than that which you have gained from your text-books and lectures. It is probably true that a large proportion of medical students have never, during the whole of their tutelage, seen a patient suffering from an attack of asthma, and meet with it for the first time when they take up the duties of private practitioners.

The attack, however, is so graphically described in your text-books that probably most of you could at the present moment give a fair account of the attack, not omitting a description of the way in which the patient will even break the panes of glass of the windows in order to obtain air. The fact remains, however, that clear as may be your descriptions on paper, you will make many mistakes on the one hand in overlooking the existence of asthma, and on the other in diagnosing as asthma what is quite a different malady.

What is meant by the term "asthma."—Asthma is a term which should strictly be limited to those conditions in which breathing becomes difficult in attacks; air cannot be as readily inspired nor as readily expired as usual. The attacks may last for a few minutes or they may last several hours; the attacks may come on at regular intervals of a few days or at very irregular intervals separated by weeks. The term "asthma" should be qualified as "bronchial asthma" or "spasmodic asthma": but our habit is to use the term "asthma" simply, except perhaps in the case of writing examination papers, or when we wish to refer to the asthmatoid seizures which occur as part of other diseases, when we use such terms as "renal" asthma, "cardiac" asthma, etc.

The cause of "bronchial asthma."—Despite all that has been written on the subject, little has been done in discovering the cause, and for one very good reason, that a patient suffering from asthma never dies of it; if he has suffered from asthma

even recently, and dies of some other affection, nothing, so past experience shows, can be learnt by post-mortem study of the cause of asthma. It is a most singular malady, for when it attacks an individual it may cause the most severe symptoms which terrify the patient, especially in the first attack, and which cause the inexperienced practitioner the greatest anxiety, more especially if, in his case, too, it is the first time he has seen such a thing. A malady, then, of which we do not know the cause and which presents such an alarming picture, must appeal to us acutely from every point of view.

An attack. — The patient who has already suffered has warnings of what is coming. As you know, many of the attacks come on in the night or early morning. No doubt the reason for this is readily found, though not necessarily correctly, by the fact that in the sleeping hours the patient does not accommodate himself to the altered condition of his respiratory apparatus: he does not use the artifices which will circumvent the onset of the attack which he would use if he were awake; moreover, overloading the stomach, as by a late meal, incommodes the respiration more in recumbency than it would were the patient up and about; possibly in some cases, too, the coldness of the atmosphere in the night hours will act as a further incentive in producing exudation from the bronchi, and so still further close their lumina, which are already closing in response to other and unknown agencies.

It is an interesting fact that the patient often knows what is coming; he feels oppressed in the chest, may feel chilly, and may have fever—an important point, may suffer from anorexia and flatulence, and may be irritable or unusually drowsy. After a few hours of repose, he wakes suddenly and feels that there is a threatening failure of respiration; the simple act of breathing is no longer easy. The chest is already almost fully expanded, and cannot be reduced in size preparatory for the next inspiration before the necessity for such inspiration has become, from want of oxygenation of the blood, a present and urgent need. Every muscle of respiration is called into aid, and to do this the more thoroughly the patient may leap from bed, and leaning forward with the hands resting on a table, use to his utmost such auxiliary muscles of inspiration as the pectorals and sterno-mastoids. Better and more oxygenated air is sought for in an adjacent passage or staircase, or at any accessible window. In slight attacks relief soon comes, but in the severe ones cyanosis appears, and this gives place to a deathly pallor, perspiration appears, which is all the more striking as the body is cold from exposure, etc. On closely observing the breathing it is seen that the normal character is reversed, for instead of inspiration being several times more prolonged than expiration, the latter is as long as, or longer than, inspiration; moreover, in most cases one or both phases of respiration are accompanied by "wheezing." Speech and coughing may be quite impossible, all available energy being required for the urgent demands of respiration. The passage of pale urine is a common feature either in the premonitory stage or in the recovery from the attack; it is almost idle to speculate why this should be in a malady the cause of which is so hidden from us, but it is possible that the hydruria is an expression of the fear produced in the patient. The heart's action may not be interfered with, but in some cases it becomes rapid and even irregular. After a time the patient's attack passes off and he may sleep, awaking much recovered, but it is a mistake to say that between different attacks he is perfectly well: he may be, but that is the exception.

It is necessary to say a few further words about the nature of the breathing in asthma. Using the term "dyspnœa" in its literal meaning—a disturbance of normal breathing—no doubt asthmatic breathing is a form of dyspnœa: but many clinicians probably use the term dyspnœa as synonymous with tachypnœa, *i. e.* hurried breathing, or to describe those frequent respiratory efforts which occur in various diseases as the *result* of extra muscular effort. By no manner of chance may asthmatic breathing be described as dyspnœa in this sense. Asthma may be said to be characterised by the occurrence of paroxysms of dyspnœa, using the letter in its literal sense, which develop independently of muscular effort, in which inspiration and expiration are prolonged and difficult of execution and in which wheeziness often also occurs.

Clinical features.—One of the most charac teristic features about the chest of a patient who has suffered from repeated attacks of asthma is that it has assumed emphysematous characteristics, the details of which are well enough known to you. But when a patient is suffering from a present

attack of asthma two cardinal points come before one. The first is that the patient's breathing is not only "bronchial" in so much that expiration is prolonged to the same or even to a greater length than inspiration, but it is also observed that the patient cannot execute either phase of respiration in the usual time; both are unusually long, and the period during which no respiration is taking place is unduly shortened. The next point is one of very considerable theoretical interest, and it is this, that many but not all attacks of asthma are accompanied by "wheeziness"; the patient and his friends may notice it, or it is only heard by the physician, who finds that inspiration and expiration, or only one of these acts, is accompanied by rhonchi, either sibilant or sonorous in type.

It is necessary to remember that asthmatic attacks may be present and yet no rhonchi be heard; this is very important, for absence of such sounds may indicate that the lumina of the bronchial tubes are not blocked by secretion. Such blockage may have been in these cases produced by mere hyperæmia of the mucous membrane or by spasm of the bronchial musculature. It is easy to understand how exudation and even mere hyperæmia may be produced on a mucous surface which is exposed to outside infection, but it is less easy to find an explanation of bronchial contraction dependent upon the calling into play of the bronchial musculature.

Another point of interest in the clinical aspect of an asthmatic attack, is that though the altered breathing is shared by all parts of the chest, the distribution of the rhonchi when they are present is by no means so regular. They may, for instance, be heard all over the lungs, but it is not unusual for them to be heard at the front or back of the chest only, or over one lung only, or over one lobe or smaller part of one lung, not only at a base, but even at an apex. Such facts as these point to an agency which operates on all bronchial muscles, but which is capable of provoking moist sounds in but a limited part of the lungs.

The localisation of the anatomical point of attack in the development of asthma.—It is quite in keeping with the history of medical studies that for a long time we have satisfied ourselves with a study of the localisation of the particular point at which the cause or causes operate. The study of asthma is no exception to the rule laid down by our medical ancestors, that our first duty is to localise, and then if possible to detect the cause.

The act of breathing is one of those functions which is volitional on occasion, but in the greater part of existence is a pure reflex, and it thus follows that investigations in asthma have been directed towards discovering whether the point of attack is in the afferent tract, at the respiratory centre, or at the efferent tract. All the more practical explanations of an asthmatic seizure are based upon the idea that it is at the periphery of the reflex arc that the disturbing factor operates; but to this day it is not decided whether the point is at the periphery of the afferent nerves leaving the mucosa of the bronchi, etc., or whether it is at the periphery of the efferent nerves which are subservient to the contraction of the involuntary muscular fibres of the smaller vessels supplying the bronchial mucosa, or to the contraction of the involuntary muscle-fibres first described by Reisseissen as existing in the trachea, bronchi, and bronchioles. The question may also be raised whether the muscle-fibres of the bronchi are themselves capable of being the seat of attack. So far as we know at present no experiments or possible deductions have been made pointing to the possibility that the muscle-fibres are the seat of attack. The question of localisation, therefore, may be confined to the consideration whether the cause or causes operate in the terminations of the sensory or motor nerve of the mucosa.

So far as the sensory nerve-endings are concerned, despite the immense amount of clinical evidence which supports the view that asthmatic seizures may be induced by irritation of various sensory nerves, there is very little proof experimentally that such may occur, and this is due to the fact that no method of investigation has been devised by which an animal may be prepared for experiment without at the same time interfering with the activity of the afferent nerves such as those of the skin, stomach or intestines. Drs. Brodie and Dixon have, however, succeeded in showing that stimulation of the nasal mucous membrane in decerebrate animals produces a constriction of the bronchial tubes of gradual and prolonged course.

Much more satisfactory proofs of the share that may be taken by the bronchial musculature and by

the peripheral motor-nerve endings in producing contraction of the air-tubes, etc., have been obtained. It is interesting to refer to the fact that the first experiments supporting this idea were carried out in 1840 by Dr. C. D. B. Williams,* a former Professor of Medicine of University College and physician at this hospital. He was able to show that in pithed animals, it was possible to demonstrate by inserting a manometer in the air-passages, that air could be driven out of the lung tissues by galvanic stimulation of the lung substance, trachea, etc., and this he attributed to the stimulation of the muscular fibres present in the walls of the air-passages. He was not, however, successful in demonstrating that the stimulus passed through the vagi. It has, however, been possible to two later observers, Drs. Brodie and Dixon, to prove that by using more delicate instruments, such as the oncometer, stimulation of the vagi can produce a marked diminution of the air contents of the lung. The vagi thus are the motor nerves of the bronchial musculature.

So far as experiment goes, then, it may be said that constriction of the bronchial musculature may occur by irritation of the motor nerves, it is also strongly suggested by experiment that such contraction may be brought about by stimulation of certain sensory fibres of the nasal mucosa.

These facts are important from our point of study, because they indicate that in true spasmodic asthma the operating agent or agents may act in the walls of the trachea, bronchi, or bronchioles, and they may act by producing a contraction of the involuntary muscle-fibres which exist in these localities either directly or through the motor nerves.

If we turn to the study of the noxa which are capable of acting in these localities we are without any solid facts. All that we know is that these surfaces are open to the atmospheric air, and that, therefore, outside agents such as dust, pollen, gases, and those commonest of all agents of disease, bacteria, have ready access. Such agents may operate on the nerve-endings directly with or without producing cartarrhal conditions of the mucosa, and may therefore produce one or other of the two clinical forms of primary spasmodic asthma, viz. that form in which dyspnœa occurs alone or that form in which dyspnœa is accompanied by catarrhal changes of the bronchial mucosa as evidenced by the production of "wheeziness" or sibilus formation.

These points will prepare us for the mixed results which treatment of the malady unfortunately bring before us, for signal and real success in some cases and for as completely ineffectual ones in others.

The clinical differentiation of asthmatic affections.—In no malady is more care required to clear the ground by a searching history than in cases of periodic dyspnœa, and I do not think any plan is better than the one which proceeds on an anatomical basis; it is an easy one and it is eminently practical.

Before making a diagnosis of true spasmodic or bronchial asthma, it is necessary to assure yourselves that you are not accepting the patient's notion of what is asthma; a positive answer must be obtained to the question, Does the patient suffer from *attacks* of difficulty in drawing the breath in and of expelling it? Once you are assured that this is the case then proceed on the plan of first of all excluding conditions in which such breathing does occur but is secondary to some other cause. Exclude these and then probably the case before you will prove to be one of true spasmodic or bronchial asthma, an "idiopathic" asthma the cause of which is quite unknown to us, but the treatment of which may be very successful.

(1) *Enlargement of the tonsils and adenoids.*—This condition is one of the most fruitful causes of error. It is the condition which accounts for so many of the cases known to our forefathers as croup, or speaking in modern language, of non-diphtheritic croup. Remove the cause and such a case of "asthma" will be cured. Our London hospitals are just now being flooded with cases of "tuberculosis in children," or "asthma in children." Routine examination of the tonsils and naso-pharynxes of these children would soon clear up the diagnosis, and a timely operation will do the rest.

(2) *Nasal disorders.*—Cases will come before you labelled "asthma," which are entirely dependent upon the existence of nasal polypi, deflected septa, hypertrophic rhinitis. Very frequently the patient, an adult one, will have attacks of so-called asthma, which alternate or are associated with attacks of repeated sneezing. In all these cases attention to

* 'The Pathology and Diagnosis of Diseases of the Chest,' 4th edition (1840), p. 320.

the nose should be carried out, and though success will not always be obtained in the treatment of the group of cases in which sneezing is so marked a feature, yet a considerable measure of success has been obtained in the others by local treatment of the nose. None of them are cases of true asthma, and include the cases sometimes spoken of as "hay-fever."

(3) *Infection of the respiratory passages.*—It is possibly true that many cases of apparently real asthma would never occur if infection by the infiuenza or other micro-organisms could be avoided. There can be little doubt that such infection may, by causing swelling of the bronchial mucosa, so impede the passage of air as to provoke a condition of asthma. Indeed, it is arguable that every case of true asthma is really due to infection of the respiratory tract, the absence of fever, of catarrh, and of a longer latent interval than occurs in some cases of asthma notwithstanding. There is every encouragement for the belief that just as so much of the disturbance of the healthy organs in other conditions is due to infection, so in the case of true asthma a similar cause may be looked for. Contemporary medicine, however, is not yet enamoured of this view, though there are signs that it would coquette with the notion.

(4) *Thymic asthma, laryngismus stridulus or child-crowing.*—These terms are synonymous for a peculiar condition occurring in children, especially rickety ones, in which there are evident defects of breathing, in which noises are produced simulating crowing. That an enlarged thymus is responsible is not accepted by most English writers, but that an irritable condition of the nervo-muscular mechanism of the whole or part of the respiratory tubes underlies it is not improbable, the actual cause of which is quite unknown. Such cases have little in common with true asthma.

(5) *Symptomatic asthma (asthmatoid attacks).*—The history obtained from the patient may suggest that he is suffering from real asthmatic attacks. The necessity for a thorough present state again comes in, for such symptom may occur as part of the clinical picture of cases of (*a*) dilated aorta, or aneurysm, or mediastinal tumour; (*b*) morbus cordis, the so-called "cardiac" asthma; (*c*) a stomach dilated by food or air, so-called "peptic" asthma; (*d*) renal disease, the so-called "renal" asthma, associated or not with the sudden development of œdema of the lungs or of one lung, or of part of one lung, or upon the slower development of pleural effusion. No doubt some cases of "gouty" asthma may be included in this category, for the association of tophaceous gout and sclerosis of the kidney is a familiar one. (*e*) Emphysema. In such a case an infection of the respiratory passages would produce a picture very like asthma. (*f*) Tuberculosis. In tuberculosis of the lungs the onset of a second bronchial infection would also produce similitudes of asthma.

(6) *Hysterical or nervous asthma.*—All are familiar with the story of the young lady who had asthmatic seizures if she smelt a rose, even when the rose was an artificial one. Hysterical patients will feign an attack of asthma, and you are probably aware of the out-patient here who in ignorance of the clinical features of tuberculosis feigned those of asthma because her tuberculo-opsonic index varied from ·9 to ·92 after massage and exercise.

(7) Somewhat allied to the last group are those singular attacks of asthma which are produced by ipecacuanha powder, pollen of grass, dust, scents or odours, as of sweat; of various animals, as of the rabbit, cat, dog, or horse; of privet hedge, or of a feather bed, or of a poulterer's shop.

One of my earliest recollections of asthma was that occurring in a guest of the family who was no mean musician and mathematician and very highly strung. A boy-like curiosity tempted me to discover in the morning how my friend had slept. I found him fast asleep after a very disturbed night, lying on the straw paillasse whilst the best feather bed the house possessed was hurled away in a corner. A more recent experience was of a far-smelling Irishman, whose efforts to avoid asthma by a sea voyage in mid-Atlantic were doomed to failure owing to the fact "that the wind off Europe had carried the pollen of Portugal to worry him whilst afloat." A subsequent stay in Portugal itself amidst the dust and pollen of weeks of journeying failed to cause any asthma.

(8) Lastly come the cases of true asthma. Such cases appear to have no common cause, and yet the attacks are characteristic, and the above forms can be excluded.

These are the cases which may be tried with the various remedies which are known to give relief—no doubt they constitute a small residuum of the large number of cases which appear before you as

the layman's asthma. But of this I am quite sure, if you will but carefully exclude the cases which simulate true asthma, you will have more success in treating true asthma: no doubt your practice in cases of true asthma will be much smaller than that of other practitioners, but it will be more satisfactory because more likely to provide you with success in treatment.

Treatment.—(1) First, make sure that the case you have to deal with is really one of true asthma.

(2) Then lay your plans according as to whether you are dealing with a present acute attack, or whether your patient is one in whom the acute attack is over, and he is anxious to prevent a renewal of the seizures and at the moment is merely suffering from bearable, but yet uncomfortable, inter-paroxysmal shortness of breath. In the attack itself great relief may be given by light anæsthesia, using chloroform by choice rather than ether, as the latter agent may provoke bronchial exudation, which might intensify the bronchitic condition, and so provoke one embarrassment whilst relieving another. An hypodermic injection of morphia may be a more convenient remedy and is very effectual. It is very interesting that amyl nitrite or a tablet of nitro-glycerine will be equally efficacious, probably because of the well-known effects of the nitro-compounds in releasing the spasm occurring in involuntary musculatures. The use of various powders, which when ignited give off nitro-compounds, is very common, and you should at least be able to prescribe one, such as the Brompton Hospital powder of 4 drm. of stramonium leaves, 2 drm. of potassium nitrate, and 2 drm. of anise seeds. Such powders may fail, and then burning blotting-paper containing as little animal fibre, such as wool, as possible, but permeated with a saturated solution of potassium nitrate may give the desired relief. In a few cases no relief will come from these measures, and there should then be no hesitation in performing venesection, removing ten to twelve ounces from the median basilic vein.

The treatment between the attacks should aim (1) at relieving over-distension of the stomach by abstention from carbohydrate food, and (2) at endeavouring to loosen the exudation in the bronchi by the use of potassium iodide gr. iii–x, combined with potassium bicarbonate, ammonium carbonate, and camphor water; should symptoms of iodism supervene, then the mixture should be replaced by one containing ammonium chloride (gr. xx) combined with liquid extract of liquorice (3j), a little glycerine, and water. A very serviceable mixture is one which contains extract of stramonium and potassium iodide, but it seems that many people soon experience an unplesant dryness of the mouth, so that it is wise not to give more than $\frac{1}{10}$ to $\frac{1}{12}$ gr. of the stramonium extract, at least to begin with. When the above remedies fail, recourse should be taken to the use of liq. arsenicalis and the ethereal extract of lobelia; both have been found of service, though this is not my experience.

If the above plan is followed there will be less dissatisfaction with the therapeutics of asthma, but it must not be felt that all difficulties will be resolved, for there will still be cases which defy treatment, and, as already hinted, once true asthma has set in, the patient rarely is completely cured. Investigations into the infective nature of true asthma are much needed.

May 16th, 1910.

Gall-stone Diabetes.—Carr urges that the occurrence of sugar in the urine should lead one to examine very carefully into the condition of the bile-ducts and pancreas. In his case the diabetes was unquestionably due to a pancreatitis caused by pressure of a large gall-stone. The symptoms of gall-stone disease were typical, but antedated the operation only three months. At the operation the adhesions about the gall-bladder seemed to be at least a year or two old. There were many old and firm adhesions around the gall-bladder, especially between it and the greater omentum. The gall-bladder was distended with about eight ounces of rather pale, ropy bile, and a single large gall-stone about the size and shape of an English walnut was found in the common duct. The head of the pancreas was slightly enlarged and somewhat harder than the body of the organ. Careful search was made for other causes of obstruction, but none were found. Bile did not flow into the gall-bladder for some time, but did flow pretty freely before the wound was closed. The stone was evidently formed by the amalgamation of a number of smaller stones, and must have required a long time to form and to work down into the common duct.—*Journ. A. M. A.*, vol. liv, No. 18.

PITUITARY EXTRACT AND SOME OF ITS EFFECTS.*

By LEONARD WILLIAMS, M.D., M.R.C.P.,
Physician to the French Hospital; Assistant Physician to the Metropolitan Hospital.

We do not know very much about the pituitary body. By one school in our student days we were taught to regard it much as the appendix is by some still confidently, but certainly mistakenly, regarded, namely as a useless and rather troublesome heritage from our simian ancestors. By another we were warned that, as the material envelope of the soul we should be cautious, reverential, and not unduly curious in the attitude which we adopted towards it. In our own day both these views have happily become discredited, for it has been demonstrated that the pituitary gland has an internal secretion which is essential to the economy. Inasmuch, however, as its activities have hitherto seemed to be associated mainly with such conditions as acromegaly and gigantism, which are not only rare but essentially fearsome and awe-inspiring, the circumstances have not been favourable to a rational inquiry into the physiological and therapeutical aspects of the matter. The work has nevertheless been going steadily, if silently and somewhat disjointedly, forward, and I am going to ask you to bear with me this evening while I endeavour to draw aside the veil of mystery and inaccessibility which has hitherto enshrouded the question so that we may extract from the exposure what there is in connection with the hypothesis cerebri which is of practical service in our every-day work.

To those who are interested in the latest views on the anatomy and development of the pituitary body I would recommend the perusal of an excellent paper by Dr. Blair Bell, which appeared in the 'British Medical Journal' of December 4th, 1909, a paper to which I desire at the outset to express my indebtedness. Other papers which may be consulted with great advantage are those by Dr. Harvey Cushing,† Dr. Campbell Thomson,‡ and the recently issued volume of Dr. Arthur De Lille, of Paris,* which gives a very exhaustive summary of most of the work which has been done on the subject, in connection with which there is an admirable leading article in a recent number of 'The Lancet.'†

Perhaps the most interesting function of the pituitary secretion is that which it exercises in the direction of causing the contraction of involuntary or unstriped muscular fibre. If we consider the very important part which this form of muscle plays in the body, it is easy to realise not only the far-reaching physiological effects of the pituitary secretion but also the special application of which its artificial administration in therapeutics is capable. We know that the work of the circulatory system is practically dependent upon this variety of muscular fibre. The tunica media of the blood-vessels is composed of it, and it is not necessary to remind you that the walls of the smaller arteries and arterioles consist almost entirely of tunica media. It would be possible to include even the heart itself, but lest I should be reminded that the muscular substance of the heart differs histologically from that in the media of the vessels, I am willing to sacrifice this organ to the wolves of theoretical hyper-criticism, with the remark that whether or not the myocardium can be properly described as consisting of unstriped muscular tissue, the pituitary gland includes it under its sphere of influence, for it is certain that, directly or indirectly, the pituitary secretion stimulates its contractile power.

Now, if the media of the smaller arteries and arterioles are stimulated at the same time as the myocardium, it is obvious that the clinical result will be a very decided rise in blood-pressure. Such, then, is the first effect of pituitary extract to which I would ask your attention. Having regard to the fact that many of us have for years past been labouring to discover something which would afford a simple means of reducing blood-pressure, it is surely small comfort to be told of yet another drug whose only effect is to raise it. But I would ask you for a moment to turn aside from the admittedly very real dangers which lurk behind the seeming well-being of sustained hypertension, and consent to consider the other side of the picture. Hypotension or unduly low blood-pressure, though neither so inexor-

* Delivered before the Æsculapian Society.

† 'Journ. of Amer. Med. Assoc.,' July 24th, 1909, p. 249.

‡ Clinical Journal, March 23rd, 1910.

* "L'Hypophyse et la Medication Hypophysaire," Dr. Arthur De Lille (Paris, Steinheil, 1909).

† 'The Lancet,' February 5th, 1910, p. 381.

able in its activities nor so dramatic in its possibilities as its opposite, is nevertheless a condition which, from the physical lassitude and mental misery which it induces, is very well worthy of our most serious therapeutic efforts. And although we are, in strychnine, digitalis, belladonna, and adrenalin, already well provided with means of raising blood-pressure, it is only necessary to enumerate such means in order to display the various and decided drawbacks which they present to any scheme of sustained exhibition. For with low blood-pressure, as with high blood-pressure, if our intervention is to be profitable it must be sustained. It would be possible to illustrate this by reference to many morbid conditions, but I must content myself by instancing three of every-day occurrence. They are—typhoid fever, tuberculosis, and post-influenzal cardiac debility.

Of typhoid fever one would say at once that it was not, theoretically at any rate, a disease in which we should be inclined to try the effects of a hypertensive drug which was endowed with the concomitant power of increasing peristalsis. The three major complications of the disease are perforation, hæmorrhage, and diarrhœa, complications of which such a drug would certainly seem to increase the probability. Nevertheless the clinical fact remains that of all the acute conditions in which it has been tried, enteric fever is the one in which pituitary extract has shown the best results. The poison of typhoid is essentially vaso-dilative in its action. Janeway* says : "The fall in pressure begins in the first or second week and goes hand in hand with the development of the toxæmia." To combat this fall in pressure would seem to be a very important indication, the neglect of which may make all the difference not only between death and recovery, but also between prompt and tardy convalescence. It has been suggested that such good effects as could be claimed for the cold-bath treatment were due, not so much to the reduction of the febrile temperature, as to the raising of the unduly low blood-pressure. However that may be, and whatever be the modus operandi, from De Lille's work† it is abundantly clear that in pituitary extract we possess a drug which is capable of doing yeoman's service in enteric fever. At the same time as it maintains the blood-pressure at a suitable level, it promotes sleep, and, in all probability, it counteracts the toxæmia.

Pulmonary tuberculosis is of all others the disease in which a low blood-pressure is a marked and prominent symptom. It attends, and goes far to explain the languor and debility, the quick pulse, the restlessness and the insomnia. The use of pituitary extract in phthisis is attended with the effects which these considerations would suggest. It does not, unfortunately, exercise any influence upon the course of the disease, but it does give rise to very considerable subjective improvement. It increases the physical energy, restores the appetite, calms the restlessness, and promotes sleep, at the same time, and in the same degree, as it raises the blood-pressure and reduces the pulse-rate. For these it cannot perhaps be claimed that they are great therapeutic triumphs, but they are undoubtedly effects which in a chronic and distressing disease a clinician is very glad to be able to command, which through the medium of pituitary extract he will in future be able to do.

The run-away heart of the post-influenzal and other toxic states is always a source of great anxiety to the medical adviser. It is often associated with a pulmonary condition which does not conform to the ordinary types either of pneumonia or bronchitis, a condition which in the absence of the run-away heart might seem unimportant enough. When, however, the two are combined they produce a state of matters in which the prognosis is, to say the least of it, anxious. To the experienced eye the picture is characteristic : the flushed face, with its bright, searching, almost critical eye, the rapid but not distressful breathing, and the mental condition which is best described as one of abnormal eagerness. The answer is rapped out, clearly and concisely, almost before the last syllable of the question is uttered. The pulse is rapid, often reaching 130, and the blood-pressure low, not seldom 100 mm. Hg. In a condition of this kind, and in lesser degrees of post-toxæmic cardiac debility, pituitary extract is more serviceable than any drug with which I am acquainted. It seems to restore tone to the cardio-vascular system in a way that nothing else does, and inasmuch as at the same time it induces sleep it is doubly valuable. It is certainly much more efficacious than digitalis, strophanthus, strychnia, and the other classical

* 'The Clinical Study of Blood-Pressure.' (Appleton, 1904.)

† *Op. cit.*, p. 137 *et seq.*

cardiac tonics. Curiously enough, however, it cannot be regarded as, in any degree, a competitor with these others in cases of true organic disease of the heart. Where the myocardium and the tunica media are poisoned, pituitary extract is invaluable; where the myocardium is exhausted in its efforts to compensate for a valvular defect, the remedy is practically inert. There are experimental facts which strongly suggest that the pituitary secretion is an antiseptic, and certainly its rather curious behaviour in the matter of myocardial troubles is difficult to explain on any other hypothesis.

But the power of the pituitary to cause contraction of the unstriped muscular tissue in the cardio-vascular system finds its most useful therapeutic application in a condition of which nowadays I see very little, namely, surgical shock. This is the condition in which, owing to medullary exhaustion, the peripheral vessels become so widely dilated that the heart is unable to maintain the circulation. A distinguished American surgeon, Crile, invented an ingenious device in which he encased his collapsed patient, and by distending it with atmospheric air he subjected the whole surface of the body to considerable pressure, to the end that the peripheral vessels might be sufficiently supported to permit of the central organ maintaining the circulation. The device was, I understand, very effective, but it had the obvious demerits of being cumbersome and uncomfortable. Now, one of the advantages of pituitary extract is that it acts as a convenient substitute for this device, for it will contract the vessels and keep them contracted for a period of about twelve hours. May I say, in passing, that the common practice of giving strychnine in surgical shock is very much to be deprecated. Strychnine acts by stimulating the medulla, and the essential factor in shock is medullary exhaustion. To stimulate an already over-taxed organ is to court the very disaster which we desire above all things to avert.

As I have dared to invade the domain of the surgeon, you will forgive me if I penetrate still further into his territory in order to call attention to another condition in which the power of the pituitary over unstriped muscle has been turned to brilliant account, more especially by Blair Bell. Every surgeon, as a matter of course, and many a general practitioner, as a matter either of necessity or of vanity, now opens the abdomen. Setting aside the question of sepsis, which never occurs to the surgeon, and to the operating general practitioner not more frequently than one would expect, the after-treatment of these cases is rendered difficult and anxious more often by intestinal paresis than by anything else. Now, Blair Bell from experimental evidence concluded that this post-operative intestinal paresis would be favourably influenced by pituitary extract, and he induced his surgical colleagues to give the remedy a trial, with the result that his anticipations were fully realised. It is therefore hardly too much to say that the post-operative terror of intestinal paresis need no longer beset the mind of him who, whether justifiably or otherwise, goes down to the peritoneum in rubber gloves.

If all I read on the subject be correct, it is now necessary to include in the surgeon's sphere of influence the condition known as chronic constipation. At any rate, such is the claim of my friend Mr. Arbuthnot Lane, whose originality and operative skill have led him to practise removal of the whole of the large intestine in some cases of this kind where the mere physician has failed to give relief. It is to be feared, however, that the advent of the pituitary, combined with agar-agar, will henceforth baulk him of his prey. For the pituitary's action in stimulating peristalsis, combined with the "ballast" producing satisfactions of agar-agar, form a combination which no ordinary colon could possibly withstand. It is the application of the principle of the pop-gun to therapeutics.

We have already seen that the action of the pituitary upon the unstriped muscle of the cardio-vascular system results in a general vaso-constriction. Now this vaso-constriction is selective in its behaviour. In some areas it is relatively persistent, in others it is very transitory. In the kidney, for example, the initial vaso-constriction very shortly gives place to a pronounced vaso-dilation with the production of abundant diuresis. I hoped and believed that this fact would provide us with an additional weapon—of which we are certainly in need—in combating œdema of cardiac origin. I was, however, disappointed. I had not then realised what I have since been forced to recognise, namely, that the pituitary has no sympathy whatever with organic affections of the heart. The presence of a crippled or puckered valve excites in it, not, as we should expect, a tearful sympathy,

but a lofty and high-nosed indifference. Endowed as it is with extraordinary powers in strengthening the ventricular systole, powers which it puts forth with dignity, ease, and force, where a toxæmia is concerned, it nevertheless declines to lend any support to a myocardium which is battling unsuccessfully with a mechanical disadvantage. And similarly; gifted though it is with very exceptional capacity for increasing the urinary output, it remains frigidly and obstinately inert when appealed to in a case of failing compensation.

But the special vaso-constrictive effect of pituitary extract is most conspicuously displayed in its relation to the thyroid. If the hypophysis cerebri may be said to view cardiac valvular distress with haughty disdain, its attitude towards the thyroid may fairly be described as one of fierce and uncompromising hostility. For it does its best to smother the latter. The thyroid stimulates secretion from the nasal mucosa; the pituitary brings about a vaso-constriction of this mucosa which is just as pronounced and much more durable than the effect produced by adrenalin. The thyroid induces mental alertness, amounting, in large doses, to restlessness and excitement; the pituitary produces somnolence, and is, with certain qualifications, one of the best remedies for sleeplessness at our command. The thyroid fixes the salts of calcium in the body; the pituitary aids and abets the capricious ovary in discharging these salts as quickly as possible.

It is, however, by reference to Graves' disease that the antagonism between the two becomes most apparent. Exophthalmic goitre is, as you are aware, regarded by many as due to hyperactivity of the thyroid secretion. That such is not the whole explanation of its pathology is, I submit, sufficiently shown by the fact that the exhibition of thyroid extract in excess has never produced the complete picture of the disease. Many of the symptoms can be so elicited; the emaciation, the tachycardia, the restlessness, the diarrhœa, the perspirations, and even the tremors are all very common in thyroid intoxication; but no one has ever witnessed the experimental production of either of the two salient symptoms, the exophthalmos and the goitre, from which the condition in this country derives its name. It is, therefore, obvious that in Graves' disease there is not only an excess, but also a perversion of the thyroid secretion, completely to antagonise which it would be necessary to have not only an excess, but also a perversion of the pituitary secretion.

Pituitary extract nevertheless does produce a very noticeable effect upon some of the most troublesome symptoms of the disease. It calms the excitement and promotes sleep; it slows the heart and raises the blood-pressure; it checks the perspirations and prevents the sudden flashes of heat; it stops emaciation, and, finally, it modifies the tremors to the point, in slight cases, of completely abolishing them. Now, although the exophthalmos itself is seldom influenced, and the goitre never, the above results are well worthy of attainment in a disease so complicated and so baffling as this, even though it be true, as it unfortunately is, that the symptoms reappear very soon after cessation of the treatment.

The effect upon the tremors is occasionally very striking, and is particularly interesting because the same calming effect is produced by the drug upon tremors of apparently different origin, namely, those of Parkinson's disease, many of the other symptoms of which are at the same time very definitely improved by the drug. Apart from the tremors, and the muscular weakness and rigidity, the most prominent symptoms of paralysis agitans are low blood-pressure, insomnia, and perspiration, together with an intolerance of heat so pronounced that patients will often be content with a single sheet for a covering on the coldest of winter nights. Here, again, the exhibition of pituitary extract brings about a rapid and decided improvement in these secondary symptoms, while leaving the essential features of the disease apparently untouched.

In the pelvic basin of women there is a pear-shaped organ whose Greek name of "hysteros" has provided us with a peg on which to hang much that is loose in diagnosis, and much more that is cruel and harmful in treatment. Anatomically it is a far cry from the hypophysis cerebri to the uterus, but physiologically and therapeutically they are closely allied. Indeed, if under the term "uterus" we include the ovary and its internal secretion, it would scarcely be too much to say that the alliance is as close and as interesting, and, I may add, as elusive as any which is to be found in the body. For the stimulating effect of pituitary extract upon unstriped muscle is nowhere more pronounced or more striking than it is upon the muscular tissue of the womb. Upon this subject Blair Bell's paper is particularly full and instructive. He tells

how, after a Cæsarian section, he gave a single injection of pituitary extract, and immediately the enlarged, congested, and distorted organ became, as he expresses it, "a blanched ball" under his very eyes. He goes on to show the powerful influence which the drug possesses upon the uterus at all times, but more especially upon the uterus in the pregnant, the puerperal, and the menstruating state. I will not stop to emphasise the immense importance of these facts to those engaged in general practice, but will proceed at once to the consideration of some practical details which I have purposely kept for the close of this rather discursive paper.

Developmentally, there is a communication between the hypophysis cerebri and the alimentary tract. It was therefore assumed that the administration of pituitary extract by the mouth would, as in the case of the thyroid, be followed by the production of its physiological effects. This has to some extent proved to be correct, bnt there can be no question in my own mind that the intra-muscular route is the more effective. At any rate, in cases in which pituitary extract seemed to be indicated, I would not consider that the remedy had been given a full trial until it had been administered by subcutaneous injection. The dose of the extract is usually quoted as from two to five grains; but there is, with one reservation, no danger in employing much larger doses, for the drug seems to be practically devoid of any effects more serious than undue somnolence and troublesome polyuria. The reservation just mentioned is nevertheless a very important one. From what has already been said as to the hypertensive power of pituitary extract, it is obvious that when exhibiting the drug for what purpose soever, it is essential that an accurate and watchful eye should be kept upon the state of the blood-pressure. In cases of sustained high tension the drug must not be used at all, and even where the tension is initially normal or even subnormal, its prolonged use is permissible only when associated with daily estimation of the state of the blood-pressure. And here let me repeat the oft-reiterated warning against the ridiculous idea which seems still to survive in some benighted quarters that blood-pressure estimation can be efficiently performed by the unaided finger. Such a claim would be ridiculous were it not so dangerous. And dangerous as it is under ordinary circumstances, that danger is enhanced an hundredfold when the use of so powerfully hypertensive a drug as pituitary extract is in question. The man who will not use an instrument to estimate blood-pressure ought not to be allowed to prescribe pituitary extract. To attempt that estimation by fingering the radial pulse is as senseless and as futile a procedure as would be the fingering of the anæsthetised eyeball to gauge the degree of myopia.

May 16th, 1910.

EPSOM COLLEGE.

It is interesting to note how fully and worthily those responsible for the development of Epsom College have fulfilled the onerous duties entrusted to them, and the medical profession has reason to be proud of the successful and prosperous maintenance of the collegiate institution on the Epsom Downs, where over 287 boys receive an excellent education, sufficiently definite in its range to ensure against diffuseness, but adequately broad enough to prevent undue specialism. In the course of the speeches delivered on Founder's Day last year, the appropriate occasion was utilised by the orators to congratulate Epsom College on the honour of the baronetcy conferred on the Treasurer of the College, Sir Henry Morris, and it is known to all concerned in the affairs of the institution how much the College is indebted to Sir Henry for labour bestowed without stint on its affairs, and for watchful care given to its financial matters. The difficult problem of combining scientific work with classical study has been solved at Epsom in a masterly manner, and the majority of thinking men in the medical profession are satisfied that the Epsom scholars are best fitted for the work of life by following a curriculum in which, although science takes a leading part, the view that classical teaching is the best way of building up vigorous mental power is recognised and acknowledged in an ample and satisfactory manner. The catholic character of the education supplied justifies the belief that the number of scholars will soon be more than 350, and the attendance of so many boys can be anticipated with confidence, because, though special facilities exist for the preparation of students for the medical profession, the policy has been always pursued at Epsom College of broadening the education as much as possible, so that boys could enter the various professions. The school is therefore not exclusively for the sons of medical men, but boys belonging to other professional and to the higher commercial classes are also educated there. There are a certain number of "day boys," and in order to give facility for their attendance the school hours have been so arranged as to enable them to come and go by train. There are a few Exhibitioners, sons of duly qualified medical men, and there are nine medical scholarships at the hospital schools in London, ranging in value from £120 to £150, which are competed for annually.

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CONTENTS.

PAGE

***Three Clinical Lectures on the Heart.** By SAMUEL WEST, M.D., F.R.C.P. LECTURE I 97

***The Treatment of Trigeminal Neuralgia.** A discussion by Mr. JONATHAN HUTCHINSON, Dr. WILFRED HARRIS, Sir VICTOR HORSLEY, Mr. BALLANCE, Dr. LEONARD GUTHRIE, Dr. GRAINGER STEWART, and Dr. SAMUEL WEST ... 101

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THREE CLINICAL LECTURES ON THE HEART.

Delivered at St. Bartholomew's Hospital.

By SAMUEL WEST, M.D., F.R.C.P., Physician to the Hospital.

LECTURE I.

I PROPOSE in this lecture to deal with certain problems connected with the heart, and to consider how far the explanations given of them are in accord with current physiological teaching.

First in respect of **the apex-beat.** The apex-beat is felt in a particular place—the fifth intercostal space in man, within the nipple-line. When the heart contracts it shortens, and therefore if nothing else happened the heart ought to be drawn away from the chest-walls, and we might expect *à priori* if we did not know to the contrary that we should get a recession or drawing-in of the chest-wall at the apex instead of propulsion or protrusion there. Whether the apex twists and turns or not is not of importance, because as the ventricle shortens it would be further away from the chest-wall and not nearer to it when the systolic contraction took place. Then it is clear that if that be so the heart must descend when it contracts. Why does it descend? The heart is hung on the aorta, like the old clapper-bell on its spring. If the spring is straightened the bell will come lower down, and that is just what happens in the case of the heart. The heart hangs on the aorta like the bell on the spring, which is attached beyond the arch to the spine. When the blood is driven forcibly out of the heart it straightens out the aorta, with the result that the heart descends.

Now let us apply this to clinical medicine and consider some of the conditions under which alterations in the apex-beat occur. First, we can

have certain changes in the spring, or in its mode of fixation, of such a kind that it cannot be straightened. In the one case the aorta has lost its elasticity, in the other there is a mechanical impediment to its movement. Secondly, there may be some altered condition of the heart itself, which prevents the heart from driving the blood with sufficient force into the aorta to straighten out the spring. All these conditions are met with in disease.

(*a*) Take the first, though this is not the commonest, as, for example, where the aorta is markedly atheromatous and has lost all its elasticity. In such a case the impulse of the heart often cannot be felt. Yet it must not be assumed that the converse is also true, and that the absence of the apex-beat is a proof that the aorta has lost its elasticity. That, of course, would be going too far.

(*b*) Next consider the condition of the aortic valves and of the aortic orifice. In order that the aortic spring shall be straightened out, the blood must pass readily through the valves into the aorta, and that with some force, for if it only trickled through it would produce no effect on the spring. It is the sudden rush of blood that does that; and if the blood does not rush into the aorta the spring will not be straightened out, and the apex will not come down. As a fact in aortic stenosis the cardiac impulse is often absent. This may seem surprising, because aortic stenosis is one of the diseases which gives rise to a very large amount of hypertrophy of the left ventricle, and it might be expected that hypertrophy of the left ventricle would give a more forcible and not a weaker impulse. And yet in spite of the hypertrophy the impulse is often absent or hardly to be felt at all.

(*c*) Now, consider the heart itself, *i.e.* the muscle. If the blood be not driven with sufficient force into the aorta it will not straighten the spring, and if the heart muscle is weak it will contract with deficient force. So a weak heart gives a feeble impulse, and a very weak heart may give none at all. When the heart is very weak and the left ventricle much dilated the heart is very much bigger, and there may then be an extension of the impulse consequent on the dilatation.

(*d*) There is still another condition of things in which the spring will not work as it ought, viz. that in which it is fixed in an abnormal way. If adhesions are so placed about the base of the heart as to prevent the heart from moving up and down, the aorta might retain its normal elasticity and the blood be driven into it with adequate force, yet the heart could not descend. If, then, adhesions be so placed in the mediastinum that they prevent the heart from descending, the impulse beat will not be felt; but something else will occur —exactly that which would be expected if the heart shortens and does not descend—there will be not merely absence of protrusion but actual recession, or drawing in; in other words systolic recession of the apex-beat. This is given as a pathognomonic sign of adherent pericardium. But when adherent pericardium is studied at the bedside it is found that there are two sets of cases: in the one this systolic recession occurs, and in the other not. If systolic recession of the apex-beat be present adherent pericardium may be diagnosed, but the converse is not true, for it cannot be said that the patient has not adherent pericardium if systolic recession of the apex-beat be absent. All depends upon where the adhesions are: they may be very extensive and involve the whole heart, and still the heart may be more or less free to move. If, however, the adhesions be so placed at the base, even if limited to that region, that the heart cannot move down, the systole will show itself by recession of the apex.

The second point I propose to consider is the **area of cardiac dulness.**

The cardiac dulness is of very great importance, for without it neither the size of the heart nor yet of the lungs can be accurately determined.

There are only two ways in which the actual size of the heart can be ascertained. The one is by means of the X rays, and of the special apparatus known as the orthodiascope, but there is no apparatus of this kind which can be conveniently carried into the wards or to the patient. The only other way is by means of percussion. The first thing to be recognised is that the area of cardiac dulness is not the same thing as the anatomical size of the heart. I should hardly dwell on this point did I not find so much confusion in students' minds, a confusion which is increased by some of the diagrams given in elementary text-books.

The heart is a more or less globular body suspended in the chest with only a small portion of

its surface near the chest-walls. Percussion over the part which is in contact with the chest-wall yields a much impaired note. This is called the area of superficial or absolute dulness, though the percussion note is never quite dull but only more or less impaired. This corresponds anatomically with the part of the heart which is uncovered by lung. Outside this the percussion becomes more and more resonant, until it becomes merged in the general resonance of the lung. Theoretically it might be supposed that if the percussion were forcible enough it would be possible to determine exactly where the heart lay, even deep down in the thorax, but this cannot be done, nor ought it to be, because when the chest walls over the heart are percussed there are thrown into vibration not only the parts actually percussed, but the parts around as well which are in contact with the lungs and therefore resonant. The harder the percussion the wider the area thrown into vibration. Forcible percussion is unpleasant to the patient, and, in fact, more can be made out by light than by heavy percussion, and that without distressing the patient.

The area of superficial cardiac dulness is roughly triangular in shape. To describe a triangle three points are required. These three points are: (1) The fourth costal cartilage at its junction with the sternum; (2) the apex-beat, in an average adult one inch inside the left nipple line; and (3) the bottom of the sternum. A line joining these points gives the area of superficial cardiac dulness. By somewhat harder percussion a line is obtained from the apex which reaches the third left costo-sternal articulation, and that is the deep area. The right boundary cannot be percussed out beyond the left edge of the sternum. Some of the books draw this line down the middle of the sternum, but this is imaginary and cannot be made out, because when the sternum is percussed it vibrates as a whole, and being in its whole length in contact with the lung, the general pulmonary resonance masks any defect of resonance which might theoretically exist. When the cardiac dulness is enlarged to the right, the right border jumps at once from the left edge of the sternum to the right edge or beyond. The drawings given in some of the recent books are extremely misleading. I do not know whether they are X-ray shadows or how they are obtained, but certainly they are not obtained by percussion.

What conclusions can be drawn from the size and shape of the cardiac dulness? If the left ventricle be affected only, the apex is displaced downwards and outwards, appearing in the sixth space instead of the fifth, but as the rest of the heart is not enlarged the area is merely an enlarged triangle, the dulness reaching upwards a little higher than normal and going down to the apex. Now take another common form of cardiac dulness: The apex is in the fifth space, but a little outside the nipple line, and the upper border of the cardiac dulness runs along the fourth or third rib to the nipple line or outside it, and then drops almost straight down to the apex. The right border will probably be found well to the right of the sternum too. The area of dulness then obtained is roughly quadrate in shape and no longer triangular. You may conclude from such an area of cardiac dulness, first, that the left ventricle is not much affected, because the apex is still in the fifth space; secondly, that the left auricle is enlarged; and thirdly, the whole right side of the heart, ventricle, and auricle. This would be explained by there being some obstruction between the left auricle and the left ventricle—that is to say, at the mitral orifice—and this is the shape of cardiac dulness characteristic of mitral disease.

A shape of cardiac dulness somewhat like this, but rather more globular and extending farther outwards and downwards, is produced by some conditions of general dilatation of the heart, or by pericardial effusion. In the case of pericardial effusion the bag in which the heart lies is distended with fluid. It tends to take a spherical form so far as it can, but being fixed to the diaphragm it is somewhat flattened there. It has been described as pear-shaped, with the point corresponding with the heart's apex. This is said to be of diagnostic importance—the area of dulness being pear-shaped in pericardial effusion, but rather square or roundish in the case of distended heart. This is misleading. A greatly dilated heart cannot be diagnosed from pericardial effusion by the shape of the area of dulness, for in either case, whether the distending fluid be within the heart or outside the heart within the pericardium, the shape tends to be more or less globular. Smaller degrees of dilatation are easy to diagnose by means of the physical signs, but extreme dilatation is very difficult to distinguish from pericardial effusion. If a patient has an enormous pericardial effusion and is likely to die, one way to save the patient's life is to tap the pericardium. The records of cases show that mistakes have been often made. A dilated heart being taken for a pericardial effusion, the needle has been inserted in order to tap the pericardial effusion, but has instead penetrated the heart, torn it, and caused death from hæmorrhage into the pericardium. Many such cases have been recorded, and naturally the mistake has happened more often than published records show. I have more than once had cases sent in to me for tapping, whereas careful examination showed that they were obviously cases of dilated heart and not of pericardial effusion, and so, of course, they were not tapped. Percussion alone does not provide the means of diagnosing between dilatation of the heart and pericardial

effusion. If the diagnosis were not difficult such mistakes as I have mentioned would not be made.

I now turn to **the heart-sounds**, about which I have also some remarks I wish to make. Students are taught in their physiology course that there are two heart-sounds; the one thing they do not usually do is to listen to them. There really are four heart-sounds not two, two for each side, but as the two sides of a healthy heart act together, only two sounds are heard, "labb-dup." In disease those sounds are often doubled. They are not synchronous, they do not come exactly together, for there is a difference in time between the contractions on the two sides of the heart; sometimes one sound is reduplicated, sometimes the other. There may even be a double reduplication, and then four sounds are heard.

How far does the simple explanation usually given of the heart-sounds fit in with the facts of clinical observation?

Take *the second sound at the base of the heart.* This is produced largely by the closure of the valves at the base, viz. in the aorta and pulmonary artery, their sudden closure giving a sharp, clear, short sound. The second sound is heard, not at the base only, but also at the apex. And it is stated that the second sound at the apex is the second sound at the base transmitted to the apex. If that were so there ought to be a decreasing intensity of the second sound on passing from the base to the apex. As a matter of fact this is not so. The second sound may be well marked at the apex, and very faint or absent at the base, or it may be loud at the base and not audible at all at the apex, and clinical observation shows that there is no relation at all between the second sound at the base and that at the apex, but that they vary independently of each other. There must therefore be some cause for the second sound at the apex other than the transmission to the apex of the second sound from the base.

Upon this problem mitral stenosis throws some light. The physical signs of that affection are not disputed. At the apex, apart from the murmur, there is a very curious alteration in the first sound and a complete absence of the second. Instead of "labb-dup," you get "prr-lab," but no "dup." The murmur we need not discuss just now. What I want you to observe is that there is no second sound at the apex. In some stages of mitral stenosis the second sound is audible at the apex, but in a well-marked typical case it is altogether absent. At the base, however, it is distinct both over the aortic and pulmonary orifices and it may be reduplicated, while the pulmonary second sound is often greatly accentuated. If the second sound at the apex were the second sound at the base transmitted to the apex, it ought to be generally audible at the apex in mitral stenosis, yet it is generally absent. The reason of this must be sought in the morbid anatomy of mitral senosis. Mitral stenosis is a narrowing of the mitral orifice, which in the end leads to a button-hole orifice; the blood cannot pass freely through the narrowed mitral orifice as it should. Instead of jumping through, as it were, it only trickles through. Under normal conditions the blood passing quickly through the mitral orifice would impinge with some violence on the walls of the ventricle, and we might suppose that this impact could produce a sound. It is only a supposition, because it has not been proved, and I do not see how it can be proved by experiment. If blood could not impinge in that way the second sound would be absent. Mitral stenosis, therefore, suggests that though the second sound at the base is due to the closure of the valves at the base, the second sound at the apex has another cause, viz. the impinging of the blood upon the walls of the ventricle at the commencement of diastole. If this impact of blood at the commencement of diastole be the cause of the second sound at the apex, the impact at the commencement of systole ought to be a factor in the production of the first sound at the base—and I believe it probably is—but I do not think this question has been investigated.

The first sound of the heart is usually explained as being partly muscular and partly valvular. The valvular part should give rise at the mitral and tricuspid orifices to a somewhat similar short, sharp snap as at the aortic valves. There are conditions of disease in which you do get exactly that change, and in which the second sound is like the first, that is to say, the sounds are "tic-tac," "dup-dup," instead of "labb-dup," and it may be supposed that owing to weakness the muscular sound is deficient.

If, then, the sounds differ in cause and character so much in different forms of heart disease, it is evident that in order to arrive at a proper conclusion as to the exact condition of the heart in any case an elaborate and systematic examination must be made. There are four regions to be examined, viz. the aortic and pulmonary, the mitral and tricuspid—and at each of these regions there are four things to consider, viz. the two sounds, and the two intervals between them. It is not enough to know that there is a murmur here or an alteration of sound there, but all the changes both in sounds and intervals must be noted and considered in relation to each other. The observations are not difficult and need only care, but the interpretation of all the facts requires experience.

One practical conclusion is obvious: where the heart retains its sounds, the disease is likely to be much less serious than where they are absent, and the prognosis to be more favourable.

May 23rd, 1910

THE TREATMENT OF TRIGEMINAL NEURALGIA.

A DISCUSSION * BY

Mr. JONATHAN HUTCHINSON, Dr. WILFRED HARRIS, Sir VICTOR HORSLEY, Mr. BALLANCE, Dr. LEONARD GUTHRIE, Dr. GRAINGER STEWART, Dr. SAMUEL WEST.

Mr. JONATHAN HUTCHINSON: It is appropriate that this subject should be discussed at the Medical Society, since it was here that Mr. William Rose, in the Lettsomian Lectures for 1892, brought forward his operation on the Gasserian ganglion by what is best termed the pterygoid route. Mr. Rose's pioneer work appeared at the same time as Mr. Hartley's and Professor Krause's, and although most surgeons have decided in favour of the temporal route (Hartley-Krause method), it is of interest to note that the late Professor Paul Poirier and Dr. Cushing, of Baltimore, advocated and practised an operation closely allied to that introduced by Mr. Rose.

It is unnecessary here to refer to the great variety of operations on peripheral portions of the fifth nerve that have been practised; their failure to produce a permanent cure in true epileptiform neuralgia led surgeons to attack the nerve higher and more centrally, and it is the universal experience that operations on the Gasserian ganglion alone are decisive and lasting in their effects. But one method may be noted, as it resembled the one which has lately been advocated as a substitute for excision of the Gasserian ganglion, namely, the injection of alcohol into or around the main trunks concerned in the neuralgia. Injections of a solution of osmic acid were repeatedly tried, this reagent being chosen on account of its selective power for nervous tissue. I have no personal experience of the use of osmic acid in the treatment of neuralgia, but I believe it is correct to say that whilst temporary relief was secured in several cases, no real cure was ever effected in a genuine case of severe tic douloureux.

Dr. Purves Stewart ('Brit. Med. Journ.,' September 25th, 1909), writing in advocacy of Schlösser's treatment—the injection of alcohol into the main nerve-trunks—refers to "the pain, risks, and serious deformities of the major operation on the Gasserian ganglion." Excision of the ganglion "is a grave intra-cranial operation with a high mortality even in skilled hands. Even when successful it produces considerable deformity with profound anæsthesia of the face, eye, nose, and mouth, together with unilateral paralysis of the muscles of mastication, and occasionally a destructive bacillary ophthalmia in the anæsthetic eye. Moreover, in spite of all this, cases of recurrence of pain are not unknown even after gasserectomy."

Such is Dr. Purves Stewart's attack upon the surgical position, and the pessimistic view he expresses is so prevalent in the profession that many patients endure the terrible tortures of this form of neuralgia for years rather than resort to an operation which they are warned may have such grave results.

I have thought it best to bring forward my own personal experience of the operation, and to state with due candour the exact results obtained. I have operated on the Gasserian ganglion by the temporal route thirty-one times, and will now discuss the results.

First as to the mortality of the operation. Is it really "enormous," as Prof. Chipault, the well-known neurologist and surgeon of Paris, described it in 1902? or, as Dr. Stewart states at the present time, has it still "a high mortality even in skilled hands?" It is perfectly true that considerable ground for these views might be obtained from the early published collections of cases. In 1896 Tiffany collected 108 cases, from all sources, with 24 deaths, *i. e.* about 23 per cent. Two years later Prof. W. W. Keen and Dr. W. G. Spiller published four cases of their own, two fatal from hæmorrhage and injury to the brain, whilst one of the survivors lost the eye subsequently. It would be possible, though somewhat unfair, to quote this as proving the present mortality of the operation to be 50 per cent. Of Prof. Krause's first thirty-six cases six patients died, a mortality of nearly 17 per cent. But the remarkable way the death-rate of all important operations becomes diminished after a few years have elapsed from their first introduction is well known.

In 1905 I pointed out that the mortality in a series of 140 operations on the Gasserian ganglion by Sir Victor Horsley, Professor Lexer, and myself was only 4 per cent. (*i. e.* six deaths in all). Of my

* Before the Medical Society of London.

thirty patients operated on not one died. Sir Victor Horsley, writing in September, 1905, gave his own mortality as being 7 per cent. It will, therefore, be allowed that the risk to life involved by the operation in practised hands has greatly diminished, and is now only slight, and is entirely different from the generally received 20 to 30 per cent. It compares favourably with the mortality following prostatectomy and gastro-jejunostomy for cancer of the pylorus, both operations performed in elderly subjects (the average age of my Gasserian ganglion patients has been fifty-two years). Neither of the two operations named can be compared as regards difficulty of performance with excision of the ganglion.

It should, moreover, be remembered that the mortality during the early days of several of our most valuable and safe operations was at first so grave as to render their justification difficult. Not to dwell on so familiar an instance of this as ovariotomy with its early death-rate of 80 per cent. and its present one of practically *nil*, I would quote an operation that came in about the same time as that now under discussion—the suture of perforated gastric ulcer. A solitary successful case was published in 1892; in 1894 the late Professor Mickulicz collected all the cases he could. They amounted to eighty-five operations, with eighty-four deaths. Surgeons might have contended that it was hardly worth while continuing to perform such a hopeless operation. Yet some improvements in technique and its more early performance have entirely changed the prognosis in the course of a few years, and it is now claimed that from 50 to 75 per cent. of the cases operated on within twelve hours of perforation recover.

With regard to excision of the Gasserian ganglion the risk has greatly lessened owing to the rejection of too extensive, severe, or clumsy methods of performing the operation, and to many small, but important lessons which experience has taught during the nineteen years since Mr. William Rose first attempted it (April, 1890).

It may, then, fairly be stated that the risk to life involved by the operation in practised hands is so small compared with the prospect of complete and permanent cure that it may be disregarded. It should, moreover, be remembered that in most cases the torture endured has rendered the patient's life quite intolerable.

Secondly, as to the deformity left by operation. It is impossible to avoid dividing the motor part of the third division when the ganglion is excised, and slight flattening in the temporal region follows from wasting of the muscle. This is the only "deformity" that need result, provided the surgeon limits himself to the sufficient amount of bone to be removed from the floor of the temporal fossa, provided he does not divide the zygoma, and provided he places the skin incision within the hairy scalp. How insignificant is the supposed deformity under these conditions may be judged by the fact that I have often, when showing patients, operated on some years before, to students, asked the latter to say which side has been involved, and it has been found difficult at first to decide.

The only cases out of the thirty in which any real deformity remained were three: (1) The only case in which I turned down a bone-flap by the osteo-plastic method; here a good deal of flattening of the temporal region has resulted; (2) a case in which facial paralysis on the same side has persisted; and (3) one in which recurrent hæmorrhage caused paralysis of part of the third nerve and persistent ptosis. The two latter complications will be referred to later.

I have seen a few cases in which a good deal of deformity has resulted from turning down a large flap of scalp and bone from the temporal fossa; I have only employed this method once, and would strongly advise against it. The flap of soft parts need not be more than 1½ in. wide; it should be placed entirely in the hairy scalp. A sufficient aperture should be made with trephine and cutting forceps, and no bone should be replaced.

Any fear of deformity after the operation may then be dismissed.

Thirdly, as to the resulting anæsthesia of the cheek, lips, and side of the mouth. This is an inevitable result—the payment the patient must make for the cure of his neuralgia. It is after all a very small one. Some complain of the numbness or board-like feeling, others soon cease to notice it. Invariably the patient, who may have been reduced to fluid diet and semi-starvation by the attacks of intense pain caused on mastication, can take food well after the operation. Talking and eating, two of the most important functions of life, can be once more performed in comfort. Compared with this, what signifies a limited anæsthesia of the face?

It is, however, a different question with regard to anæsthesia of the eye, which Dr. Stewart appears to regard as an invariable result of the operation. Struck with the fact that a cornea which has lost all sensation is never safe from risk of injury, and that excision of the globe has had to be performed in many cases of what may be termed the classical operation (advocated by Professor Krause, Sir Victor Horsley, Dr. Cushing, and others), I have urged a modification by which the ophthalmic division is left intact.

Experience has led me to attach great importance to this point. In four cases only I have done the usual or complete operation—in two because the neuralgia had affected all three divisions, and in two owing to failure to carry out the section of the ganglion exactly as intended. All these four patients have had more or less trouble as regards the anæsthetic cornea, in spite of every precaution with regard to the use of protective glasses, etc. In one keratitis came on some months later, and as it would not improve under treatment the eye was excised—this was perhaps premature. In the other three patients conjunctivitis or kerato-iritis, though alarming, has yielded to treatment. But in such cases there is always the risk that a little septic dust may lodge on the cornea and lead to irreparable mischief. Hence they are condemned to wear protective glasses when out-of-doors.

It will be seen that in twenty-six cases the section of the ganglion, so as to leave intact the ophthalmic division, was carried out exactly as intended. The result has been that no possible risk of corneal ulcer or other ocular complication has remained, surely a very important matter, and one that removes, to my mind, the chief objection to excision of the Gasserian ganglion.

As regards the argument that has been raised that recurrence of the neuralgia will take place in the ophthalmic division, I can only state emphatically that no single instance of this has come under observation. I have followed up my cases diligently, the earliest operation dating as far back as 1897.

It is not merely that by the modification I have urged the risk to the patient's eye is avoided, but the whole operation is made more easy to the surgeon and much less dangerous for the patient—especially as regards injury to the cavernous sinus.

Fourthly, the last objection to surgical intervention that remains to be considered is that the pain sometimes returns, or to put it in more general terms, that the operation does not always effect a complete cure. It would be difficult to name a single measure of medical or surgical treatment which is infallibly and entirely successful in effecting a cure of the disease for which it is employed.

It is certain that all forms of extra-cranial operation, however elaborate and severe, after a period of partial or complete cessation of the pain of a few months to one or two years, have ended in disappointment. The pain has returned in worse degree than before. The late Prof. Bilroth, whose experience was very large, candidly admitted that this had been invariably the case. Intra-cranial division of the second and third main branches of the nerve is no more permanent in its result. This was strikingly illustrated by one of my cases—a man who had first excision of the inferior dental nerve through a trephine hole made in the lower jaw. Then, after an interval of relief, the Gasserian ganglion was exposed, but not removed, the second and third divisions being completely cut across at the foramina ovale and rotundum. This operation was performed at St. Bartholomew's Hospital. A year later the ganglion had grown fresh nerves towards the periphery, and the neuralgia was worse than ever. The only anæsthetic part in the face was that supplied by the inferior dental nerve, which had been resected. I found a normal ganglion and removed it with complete success.

We may therefore accept as an axiom that intra-cranial section of the main trunks, however close to the ganglion, will be followed by restoration of function and return of the neuralgia. Moreover, even if the foramina ovale and rotundum were plugged after the section with metal or plaster, I see no reason why the neuralgia should not return so long as the ganglion has been left. On the other hand, there is so far no proof that a restoration of the ganglion after its resection is possible. I hoped at one time that complete excision of the superior maxillary trunk (half an inch being cut out) between the ganglion and the foramen rotundum would cure the rare cases of epileptiform neuralgia limited to the parts supplied by this nerve. In one case of this operation cessation of the pain lasted only about a year, and later I

operated on the ganglion as the neuralgia had recurred in the third division.

In another case the cure was apparently complete, but at the end of six years of immunity some twinges of pain have come on in the third division, and it is possible that the ganglion will have to be removed.

In an admirable paper on "The Functions of the Trigeminal Nerve," by Mr. H. Morriston Davies ('Brain,' part cxviii), based on fifty cases operated on by Sir Victor Horsley, allusion is made to the occasional escape of fibres of the second division during the operation, and the consequent retention of some sensation. It is in such cases that slight return of the neuralgia may be met with.

But, apart from this, experience shows that removal of the Gasserian ganglion for true epileptiform neuralgia affords as radical and permanent a cure as any operation in surgery.

Of course it does not of necessity cure the neurasthenic condition sometimes accompanying the neuralgia, nor will it prevent the development of neuralgia of the other side of the head. Prof. Krause records complete failure in the case of a very neurotic medical man on whom he operated. In two of my cases, both women, the result has been disappointing from this cause. In a given case, if the surgeon has good reason for believing his patient to be extremely neurotic or neurasthenic (apart from the well-known depressing effects of the neuralgia and the drugs taken for its relief), he will be wise not to promise an absolute cure from any form of operation.

Mention must be made of an occasional complication of the operation, of which Mr. Morriston Davies quotes examples, and of which I have seen two or three cases, namely partial paralysis of the facial nerve on the same side. This phenomenon is difficult to explain. The paralysis is usually slight and transitory, but in one of my patients it certainly lasted a long time. There was, however, reason to believe in this case that it had been coming on before the operation was performed.

As regards the danger of sepsis, this seems to be easily avoided with due care, though no complication could well be more serious. In not one of my thirty cases has it arisen. The shock of a necessarily long operation is, as a rule, far less than might be anticipated. Recovery is rapid, and the patient is rarely in bed more than a fortnight, often less.

The great difficulty of the operation is the hæmorrhage. In some it is easily controlled, but in others every minute vein and artery causes trouble and delay. Adrenalin solution, preliminary ligature of the external carotid artery, and various other measures have been tried without visible effect. In two cases only have I met with serious recurrent hæmorrhage. One happened in a comparatively young man; the operation had been completed satisfactorily, and some hours later the typical signs of meningeal extravasation came on. I opened up the wound, clearing out a large blood-clot, and the patient recovered; but some oculo-motor paralysis (chiefly ptosis of the upper lid) remained. The bleeding vessel was clearly seen to be the *upper* or distal end of the divided middle meningeal artery.

In the other case alarming hæmorrhage came on just as the wound was being closed; it was arrested by temporary plugging with gauze.

I have enumerated and laid emphasis on all the drawbacks and complications which may occur; but in conclusion I would urge that in the great majority of cases the operation is a most successful one. The risk to life is very slight and the prospect of permanent cure almost certain.

Dr. WILFRED HARRIS: For this discussion, at all events, I shall restrict my remarks on treatment for the most part to that form of neuralgia of the fifth cranial nerve which is characterised by the definiteness of the pain-area, corresponding roughly to the anatomical distribution of one or more branches of the nerve. Further, all neuralgias are definitely excluded in which there is any detectable cause of irritation of the nerve, either central or peripheral.

The pain, moreover, almost invariably comes on in severe spasms, varying in duration from half a minute to half an hour, and there may be intermissions of many months, or even of years, in which the patient is completely free from pain. With this exception the pain continues year after year, until operation of some kind, or death, brings relief. No cause has yet been fully proved for this terrible disease, though various slight changes in the Gasserian ganglion and in the peripheral nerves have been at times described. Strictly

speaking, the pathology of this disease does not come within the four corners of this discussion, and yet I must make some brief remarks upon this point, in order to explain the rationale of what is, I believe, the best treatment in the large majority of cases.

The sexes appear to be about equally affected. Thus of 265 cases, 144 were men and 121 women. There seems, however, to be a very definite predilection for the disease to attack the right side, for, of these 265 cases, 179 were on the right side and 80 on the left side, while six cases were bilateral. Thus, the right side appears to suffer more than twice as often as the left side.

The character of the pain is sometimes exactly simulated by other forms of trifacial neuralgia, particularly dental. In this latter form the localisation of the pain closely resembles that of trigeminal neuralgia, while it may, and often does, occur in spasmodic attacks, very suggestive of the more intractable disease. For example, a most severe case I saw recently of spasmodic dental neuralgia in a young lady, æt. 23 years, which had gone on for three weeks with increasing severity, in the temple, ear, and both jaws, with complete absence of pain between the spasms, proved to be due to an erupting wisdom tooth in the right lower jaw, and lancing the swollen gum over the bulging tooth at once cured the pain. The severity of the pain in dental neuralgia may occasionally be every whit as great as in trigeminal neuralgia, as is evidenced by a case I saw at St. Mary's Hospital two months ago, of a young woman who, some months previously, had been suffering such agonies of pain from her teeth that she threw herself out of the window in the endeavour to kill herself. Indeed, the resemblance between many dental neuralgias and trigeminal neuralgia affecting the second and third divisions of the nerve is forcing me to the conclusion that the painful spasms of trigeminal neuralgia are set up by a local neuritis of some terminal twig of the nerve, similar to that set up by dental caries in the nerve-filaments of the pulp-cavity of the tooth. True trigeminal neuralgia, affecting the first or ophthalmic division of the nerve alone, is much less common than of the second or third divisions, and, indeed, I have only met with one such case. Yet supra-orbital neuralgia is common enough as a reflex from errors of refraction and other similar causes, and it is especially severe as a post-influenzal neuritis. In this latter form it shows a marked periodicity, usually commencing about 10 or 11 a.m. and lasting until about 5 p.m., and it may be so severe that the pain becomes quite unbearable. Four such cases of post-influenzal supra-orbital neuralgia I have treated with injections of about ten drops of 80 per cent. alcohol into the supra-orbital nerve at the notch, and the relief from the pain was immediate and lasting in each case. My method is to feel with the finger-nail for the notch between the upper eyelid and the eyebrow, and then to mark the spot with a dermographic pencil. With the usual antiseptic precautions, a fine hypodermic needle attached to a syringe containing the alcohol is then inserted through the skin down to the bone, and the notch is felt for. This cannot probably be felt for certain by the hand holding the syringe and the needle, but the moment when the nerve is reached is announced by the patient complaining of sharp pain darting up over the forehead. A drop or two of the alcohol is then injected, and if this causes an aggravation of the same pain, the needle is correctly introduced. Drop by drop more alcohol is injected, until it ceases to cause pain. The skin of the eyebrow, forehead and scalp over the anatomical distribution of the nerve, as far back as the top of the head or even to the crown, will then be found to be insensitive to the prick of a pin. Sometimes I have found the triangle of skin internal to the notch and bounded by the hair line escape becoming anæsthetic.

The comparative frequency of trigeminal neuralgia of the second or third divisions, as compared with the first or ophthalmic division, I cannot help believing is due to a septic neuritis affecting the dental nerves, as a result of pre-existing caries, and thence spreading, in the case of the lower jaw, to the trunk of the inferior dental nerve in its bony canal. That this must be the site of the irritative process in the nerve, whatever its nature, is, I think, proved by the result of the treatment. In trigeminal neuralgia affecting the inferior dental nerve, the pain is almost invariably referred to a point in the lower jaw corresponding to the first bicuspid tooth. Sometimes there is tenderness over the mental foramen, from which the inferior dental nerve escapes just at this level on the lower jaw, and light pressure here may induce an attack of the pain. The lower lip itself, too, may be extremely sensi-

tive, so that the rubbing of a veil, or of the bed-clothes at night, may be exquisite torture. Yet destruction of the inferior dental nerve at the mental foramen by an injection of strong alcohol at this spot will not cure the neuralgia, which continues in the jaw unchecked, although the resulting anæsthesia of the lower lip and chin will improve the condition of the patient somewhat, since now these parts will bear being touched or rubbed without producing any spasms of pain. Eating and talking are, however, just as troublesome as before in starting the attacks, and it is clear that the site of the trouble causing the neuralgia in the inferior dental nerve is on the central side of the mental foramen. On the other hand, destruction of the trunk of the inferior dental nerve at the back of the lower jaw by drilling the bone, or by injecting strong alcohol into the nerve, either at the inferior dental foramen where the nerve enters the lower jaw, or else at its point of origin from the inferior maxillary nerve at the foramen ovale, will give immediate relief to the pain, even in cases which have persisted for twenty or thirty years. The site of the mischief causing the pain must therefore lie between the back of the lower jaw and the exit of the nerve from the mental foramen. Now caries of the lower canines and incisors is comparatively rare, and in my opinion the point to which the pain is always referred, about the level of the first or second bicuspids, probably indicates the site of the septic neuritis in the inferior dental nerve which is setting up the neuralgic attacks.

Similarly, in trigeminal neuralgia of the second division, the pain is usually referred to the inside of the upper jaw, and though, as in the case of the third division, the parts supplied by the terminal branches of the nerve, in this case the infra-orbital, may be extremely sensitive, so that touching the upper lip, or blowing the nose will at once start an attack, yet destruction of the infra-orbital nerve by resection or by an injection of strong alcohol at the infra-orbital foramen does not arrest the neuralgia, although the condition of the patient may be somewhat improved by the anæsthetic condition of the side of the nose, cheek and lip, enabling the patient to wash or touch the face without starting the attacks. Yet the pain will be produced on attempts at eating or talking, just as before, indicating that the trouble lies central to the infra-orbital foramen. On the other hand, destruction of the superior maxillary nerve further back, at its exit from the skull at the foramen rotundum, will cause total arrest of the neuralgia, until re-union establishing conductivity of the nerve takes place. Hence it will be sound treatment to interrupt the conducting power of the affected nerve or nerves, either by resection, or by any process which will destroy the trunk of the nerve behind the point at which the dental nerves are given off.

Conversely, in those cases in which the neuralgia is due to some central disease within the skull, such as neuritis of the ophthalmic branch from herpes zoster, tumours irritating the trunk of the fifth nerve, etc., peripheral resection of the nerve-trunks, or injection of the nerve-trunks with strong alcohol, has no effect in arresting the pain. As a rule, there is no difficulty in distinguishing such cases from true trigeminal neuralgia, even though the spasms of pain may be mainly intermittent, as in the latter disease. When the nerve-trunk of the ophthalmic division is injured by neuritis at or below the ganglion there will be, in addition to a varying degree of anæsthesia of the forehead and temple, distinct signs of scarring and glossy skin, such as the condition termed morphœa. I have treated two such cases with injections of the supra-orbital nerve with strong alcohol; one case was slight neuralgia and paræsthesiæ subsequent to herpes frontalis, and the other one of severe pain due to a chronic neuritis of the ophthalmic division, in which the appearance of the skin of the upper eyelid and forehead was almost precisely similar to that resulting from herpes frontalis, but had developed slowly, without the appearance of any rash. In neither case was the pain improved in the slightest degree; on the contrary, in one the pain was definitely aggravated. In another case, unique in my experience, the neuralgia was due to sclerosis of the descending spinal root of the fifth nerve within the medulla. It occurred in a gentleman, æt. 73 years, who has doubtless consulted several who are present here to-night. When aged 67 he had a slight stroke, without any loss of consciousness, but he complained of a sensation as "though hot flannels had been suddenly applied to his head, and as though hundreds of needles were sticking into his scalp." Two days later he became unable to swallow, and he had to be fed through an œsophageal tube for a week, and kept his bed for four months. Since then he has constantly complained

of vertigo on looking upwards, and of feeling shaky on his legs, especially on the left leg. The stroke was followed at once by slight aching around the left eye and temple, which has gradually increased since, severe flashes of pain darting underneath the left eye and temple developing about a year later. He has now complete analgesia of the left side of the face and front of the head, corresponding to the left fifth nerve distribution, while there is similar complete analgesia of the back of the right side of the head and of the right side of the neck, and the whole of the right half of the body. The case clearly corresponds exactly to those cases of thrombosis of the inferior cerebellar artery which have been published by Spiller, Kinneir Wilson, and others. So far as I know, however, none of these cases developed any neuralgic pain. The point of my mentioning this case is to illustrate my point that peripheral resection or destruction of the nerve-trunks is useless when the cause of the pain is central to the point of operation. Here the site of the disease is clearly the patch of sclerosis in the left side of the medulla, resulting from the thrombosis of the left inferior cerebellar artery. Nevertheless I decided to attempt injection of the second division of the left fifth nerve at the foramen rotundum, and I injected 1½ c.c. of 80 per cent. alcohol, causing considerably increased numbness of the cheek and upper lip. I also gave two subcutaneous injections of the alcohol outside the left outer canthus and above the temple, the two chief seats of the pain. Instead of the pain being mitigated or entirely relieved, as it practically is in true trigeminal neuralgia when the nerve-trunk is properly injected, the pain in this case was made much worse.

The diagnosis of the site of the cause of the pain is most important in estimating the probable results of any projected treatment, and it practically comes to this, that if the pain is referred along one or more branches of the nerve, and there is no trace of any anæsthesia or analgesia, or other signs of trophic impairment, such as muscular wasting or glossy skin, then the site of the cause of the pain is surely peripheral, and a careful search must be made for any source of peripheral irritation, such as a carious tooth, an unerupted and impacted wisdom tooth, etc. It is, by the way, important to remember that the pain due to a certain tooth may be referred by the patient to the other jaw on the same side, that is to say, a carious upper molar may give rise to pain felt only in the lower jaw. The pain never seems to be referred to the other side of the face. This particular point is of great importance to remember in dealing with true trigeminal neuralgia, as in a great number of cases the patient will complain of pain in both upper and lower jaws, or in the upper jaw and forehead, when the pain is really due to only one of the branches, the other being only a reflected pain. Considerable care must be exercised in discriminating on this point, and numbers of cases will be loosely described as trigeminal neuralgia of the second and third divisions, when the pain is in reality only due to one of them, and treatment by injection of the offending nerve-trunk with strong alcohol will abolish the pain completely. A good instance of this point is the following case: A lady, æt. 50 years, unmarried, for the last nine years had been subject to attacks of intense pain in both jaws and the tongue, temple, and ear on the right side. She dates the trouble from one day at lunch, when she felt, quite suddenly, a crash on the right side of her mouth as though she had broken her jaw or a tooth. This was not actually painful, but at every meal afterwards for several weeks she noticed flashes of pain in the right cheek at meal times or on talking. The pain did not go into either jaw at first. Ever since the pain has returned at intervals, her longest period of freedom being once for nearly a year. Her last attack commenced last March, and was worse than ever. She described the pain as being like fireworks playing along the side of the cheek, and then exploding like lightning, darting into both upper and lower jaw, and into the right side of the tongue. On cross-examination she declared that the pain ran along the lower jaw as far as the middle line, but did not run along the upper jaw further than the first bicuspid tooth. This fact, together with the facts that the pain attacked the tongue and the ear, decided me that probably it was the third division only of the nerve that was at fault, and that the pain in the upper jaw and cheek was really a reflected pain. I therefore injected the right foramen ovale with 80 per cent. alcohol, under chloroform, causing complete anæsthesia of the whole of the inferior maxillary nerve, and also paresis of the masseter, temporal and pterygoid muscles. This was done on September 4th last, and there has been no recur-

rence of pain whatever since. The motor weakness is not of much moment, and the chief complaint of these patients afterwards is that the lower lip and tongue feels swollen and dead, and the lower jaw wooden, while in eating they frequently lose a portion of food, which lies unsuspected in the lower cheek, perhaps for an hour or more.

The relief from the pain is so striking and immediate, with so little advantage to set against this, that even if the pain does recur after one or two years, this treatment seems preferable to any operative treatment. During the last twelve months I have seen twenty-seven cases of true trigeminal neuralgia, or tic douloureux, twenty-four of which I have treated by injections of 80 per cent. alcohol. Of the remaining five cases, three refused treatment by injection; one, a case of bilateral neuralgia, involving both second divisions of the fifth during the last forty-two years, was sufficiently relieved by butyl chloral hydrate and gelsemium, as to render injection treatment unnecessary. The remaining case, a woman, æt. 58 years, who had suffered only three months with neuralgia of the right third division, died suddenly of angina and cardiac infarction while butyl chloral was being tried. Of the twenty-four cases which I have treated by alcohol injection, twenty appear to be so far completely successful as regards entire disappearance of the pain. Two of the remaining four cases are partial failures. One of them, a lady, æt. 55 years, is a case of severe bilateral neuralgia of the third divisions of fifteen years' duration. I injected the left foramen ovale successfully under chloroform on May 21st, after which she was free of pain and very well for six weeks, when the pain recurred on the right side, which was the original side to be affected. I have made three attempts to reach the foramen ovale without success, once without general anæsthesia, and once under chloroform.* This failure, however, is not to be ascribed to the treatment; if the nerve is properly reached and injected with the alcohol, the pain infallibly ceases, and the responsibility for the failure I must claim entirely for myself. In another case of old-standing neuralgia of the left second division, the pain was arrested entirely for four months by injection of the infra-orbital foramen, under chloroform. As a rule, this foramen is fairly easy to reach with a needle, and to inject, though not nearly so easy in many cases as it appears when looking at a skull. When the pain recurred, I made two attempts under chloroform to reach the foramen rotundum and the infra-orbital foramen, but produced no return of anæsthesia, and the pain was not improved.

* Since successfully injected under chloroform, causing deep anæsthesia of inferior maxillary nerve.

My other two failures were one of the third division and one of the second division. In this latter case I found it very difficult to get my needle between the front edge of the coronoid process and the back of the antrum, so as to reach the pterygo-maxillary fissure. I did reach it, however, on three separate occasions, twice without any general anæsthetic and once under chloroform, but failed to hit off the nerve at the foramen rotundum. Indeed, in this case the alcohol injected must have found its way to the sixth nerve and even to the optic nerve, as slight diplopia was produced in certain positions of the head, and a certain degree of central scotoma for colours. In one of my successful cases, a woman, æt. 57 years, who had severe intermittent neuralgia of the right second division for sixteen years, injection of the foramen rotundum and infra-orbital foramen caused complete anæsthesia of the cheek and side of the nose, upper jaw and hard palate to the middle line, with complete cure of the pain, but some of the alcohol must have reached the oculo-motor nerve at the sphenoidal fissure, as slight diplopia and medium dilatation of the pupil were produced. The diplopia passed off after a week, but the dilatation of the pupil remained when she returned home. Pain in the ear for some weeks after injection around the foramen ovale I have seen on three occasions, with, in one of the cases, considerable impairment of aërial conduction of sounds. This I ascribe to the close relationship of the Eustachian tube to the foramen, the tube passing immediately below and internal to the inferior maxillary nerve at this point.

If the needle is pushed in as far as two and a quarter inches when searching for the foramen ovale, it is likely to pierce the wall of the pharynx, and if any alcohol is then injected the patient will recognise it by the smell, and by its burning sensation on the back of the tongue. This is of no consequence. It must be distinguished from the sensation of trickling along the lower jaw and tongue that the patient will notice when the foramen ovale is being successfully injected, owing to the destroying action of the alcohol upon the lingual and inferior dental portions of the nerve. Well-marked pruritis may be sometimes complained of in the area of nerve-distribution after a successful injection into the nerve-trunk, as in the case of a lady, æt. 37 years, who had suffered for twenty years from severe neuralgia of the right inferior dental nerve. I injected her right foramen ovale without any general anæsthetic, causing the sensations above described, besides complete anæsthesia of the whole area of the third division. For several months now she has been quite free from pain. Of my twenty-nine cases of true trigeminal neuralgia, nine were men and twenty women. Thirteen cases affected the third division only,

and eight the second division only, while only two appeared to affect all three divisions of the nerve, and in two cases each the first and second divisions and the second and third divisions were affected together, both requiring treatment. Eighteen of my cases were on the right side. nine on the left, while in two cases the pain was bilateral.

Sir VICTOR HORSLEY said that the matter of the treatment of trigeminal neuralgia was certainly, at the present time, in a very interesting stage, and he thought that valuable researches and experiences like those of Dr. Harris and Dr. Purves Stewart in this country placed the whole matter in a fairly simple way. He agreed with Mr. Hutchinson in regarding Dr. Purves Stewart's account of the removal of the Gasserian ganglion as far from convincing. He (Sir Victor) had hunted out his cases as far as he could make them out from the records of University College Hospital and Queen Square Hospital, as well as those in private practice, and found he had done the operation one hundred and forty-nine times, and in patients of all ages. He found the death-rate was exactly what was published a few years ago, and now quoted by Mr. Hutchinson, namely, 7 per cent. But in his experience that was a death-rate which applied only to patients over fifty years of age. He had never lost a patient from the operation under fifty years of age. One case was one of accidental sepsis, and occurred at University College Hospital—the only case he had had of sepsis in connection with the operation. The other deaths were due to the arterial sclerosis, that is to say, three were cases of pontine hæmorrhage, one was œdema of the brain, one œdema of the lung, and only in one case, and that in the present year, had he seen a death from shock after the operation. It was remarkable that the operation was practically never followed by shock. The association of arterial sclerosis with the condition was observed many years ago by Dana. He had been convinced of the truth of that clinical observation. There were difficulties in connection with the operation, but none of them were insurmountable. He had not done the operation in two stages for many years. As to deformity, he did not agree with Mr. Hutchinson, and he thought that the osteoplastic method was the best to avoid any external appearance. But, as he said, if the scar was kept in the region of the hair there was no visible deformity. So that both the so-called mortality and the so-called disadvantages of the operation had been undoubtedly greatly exaggerated. Then arose the question what the surgeon was going to advise the patient, and that was the practical way of looking at the matter. Since the introduction of the alcohol injection method he (Sir Victor) had gone into the matter very carefully, and had used the alcohol method with the view of avoiding the Gasserian ganglion operation. The most successful case he had had of that sort was one which he injected eight months ago, the patient being a gentleman, æt. 79 years, an extremely frail subject, and in whom he had advised no operation four years ago. He injected him freely in both the second and third divisions, and apparently, at the present moment, with a perfect result. The case alluded to of death from shock was that of a lady, a very worn out subject, in whom he advised the same procedure. He injected her second and third divisions with alcohol, but without any result. In fact the third division became distinctly aggravated, and therefore he advised her to have the Gasserian operation. She died of shock. At the operation he found that there was so much fibrous adhesion round the foramen ovale that it was certain the result could not have been different. Therefore it was an important point to consider what the surgeon should advise the patient. With regard to the class of case, he agreed with Dr. Harris that the case in which the best results were obtained by the alcohol injection method was the case in which the trouble was strictly localised to one division. Such cases were very common, and if the operator could hit off the nerve a very good result was probably insured for a time. One did not yet know how long the result was going to last. His advice to the patient was that they should try the less severe measure first. With regard to hitting off the nerve, it was difficult to hit off the foramina with the point of a needle, although one might think it was fairly simple from having seen the foramina exposed. He (Sir Victor) thought it could be perfectly well done under chloroform in every case, and his own method was as follows: The patient was lightly anæsthetised to begin with, and he introduced the needle; then he had the chloroform shut off by the tap, and the patient respired oxygen or air for four or five minutes; then when the patient was obviously coming out of the chloroform he moved the needle until he got a reflex movement on the part of the patient; from that one could see exactly when the nerve was touched, or any branches of it; and when the patient screwed up the face reflexly the injection was made. In that way one was able to avoid the disagreeables to which Dr. Harris referred, but which were inevitable when attempting to do such a painful procedure without a general anæsthetic. As he had already said, there was a class of case in which the operation of removal of the Gasserian ganglion appeared to be inadvisable for general constitutional reasons, and those one selected for the alcohol injection treatment. Lastly, there were cases in which the Gasserian ganglion operation should be approached from the first. He believed that those were the cases in which at least two of the three divisions were affected, and accompanied by physiological signs of irritation, such as hypersecretion, both of tears and saliva. And if trophic

changes were present, then *à fortiori*, even more so. But, as Dr. Harris said, each case must be dealt with on its merits. With regard to the disadvantages of removal of the ganglion, those were more a matter of imagination than clinical fact.

Mr. C. A. Ballance said that after the admirable papers on the treatment of trigeminal neuralgia to which the Society had listened and Sir Victor Horsley's speech, there was very little left for him to say. The way in which the surgical side of the subject had been approached by Mr. Jonathan Hutchinson was altogether admirable, and his paper was a model of what such a paper should be. With most of Mr. Hutchinson's conclusions he (Mr. Ballance) cordially agreed. Dr. Harris's paper also was exceedingly interesting, because it had been to a certain extent philosophical, and the author of it had approached the matter with an open mind. There could be no doubt that the dangers of the operation of removal of the Gasserian ganglion had been gradually regarded with less and less fear. Some of the operations on that ganglion were very difficult, but others were not difficult at all. He had one case which was to him very difficult some years ago, and he did not think he would have been able to complete it if it were not for the help he received from Sir Victor Horsley. With regard to what ought to be the surgical treatment of trigeminal neuralgia, he would like to mention two cases which came before him in the last two days. Yesterday he had a letter from the wife of a patient for whom he did intra-cranial division of the second and third divisions of the fifth just over three years ago. She said her husband had been having a few sharp attacks of pain during the last fortnight, and she wanted to know if it was possible for the pain to recur. That case he had operated upon three and a half years before that, doing the same operation. The pain recurred, and he opened the flap again and exposed the foramen ovale and foramen rotundum, and he believed he found a few tiny filaments of nerve passing between the divided ends of the nerves. He divided the filaments, and that patient had a complete respite for over three years. It seemed to him that if they could procure a respite from pain for over three years in certain cases without removing the ganglion, it was the better operation to do. The other case was one which he saw that morning, the wife of a doctor who had typical epileptiform neuralgia of the second and third divisions of the fifth. He asked himself how he was to treat that case; should he inject alcohol or should he remove the Gasserian ganglion, or do intra-cranial division of the second and third divisions of the fifth? He had now no doubt in his own mind that the proper course was to do injection of alcohol into the foramen ovale and foramen rotundum. He had found that operation by no means difficult. The guides which were described by Schlosser were quite adequate to find exactly the external apertures of the foramina out of which those nerves came. Therefore that was the operation which he recommended, and he was glad to find, from what Sir Victor Horsley had said, that that was probably what he would have done also. If that failed he proposed to do division intra-cranially of the second and third divisions of the fifth, and completing the operation by filling up the foramina by liquid rubber or by gold leaf. In either case he would expect there would be relief for about three years. The first case in which he injected alcohol into the foramen ovale and foramen rotundum was in an old man, æt. 70 years, who had lived a very active life. He had cardiac disease and albuminuria and was quite unfit for a severe operation. He had then only just become acquainted with the important paper about the injection of alcohol, and he therefore attempted operation with a good deal of trepidation about being able to succeed with it. But he found it comparatively easy. This patient had not been able for many years to take a meal with his children, but in the rooms of each of his houses he had had handles put up on the wall to which he might hold when the paroxysms of pain came on. It was a severe and terrible case. He injected alcohol into those two foramina, and since then the patient had suffered no pain at all. He (Mr. Ballance) was sure, therefore, that the right thing to do was to use that very simple measure at once, and then afterwards proceed to the more distinctly surgical course if necessary.

Dr. Leonard Guthrie said it seemed from the discussion that no measure was to be adopted in cases of trifacial neuralgia except some form of operative treatment. But he hoped there would be some physicians and neurologists present who would be willing to put the other side of the question, and show that it might be even possible for drugs to be of some value in such cases. One thing which had always struck him was the healthy appearance of the subjects of that condition, and one knew that trigeminal neuralgia never seemed to shorten the patient's life. It was almost unknown for a case to have proved fatal, and yet from what had been said the pain was most excruciating and of an intolerable degree. If one took the patient's own statements, one would think that they would be all subjects for having the Gasserian ganglion excised, but he did not think that was so in all cases. The complaint might in some cases be regarded more as a psychical feature than an actual pain, because most of those who suffered from acute toothache knew that if it continued even for a week they would be very much reduced in health. He had often been unable to square the complaints of the patients with tic douloureux with their general appearance. Personally he had found in the way of drugs an incom-

patible mixture, such as butyl chloral hydrate, salicylate of soda and phenacetin, in ten-grain doses, would sometimes relieve the pain a great deal. If he was correct in saying that many of those cases might rightly be termed functional, one might be able to explain why the fact of any operation being done seemed to be so satisfactory. If one could impress the patients with such an incompatible mixture as that which he had mentioned, it was natural to find that they were sometimes cured by it. On the other hand some of these patients were much more impressed by an operation, either injection of alcohol, or excision of the nerves, or of the Gasserian ganglion itself. Another important point was the age of those patients. He believed Sir Victor Horsley said he had performed the operation at all ages, but he (Dr. Guthrie) thought it was not desirable to operate on very young subjects. He would like to know at what period of age Sir Victor Horsley would suggest operation for the condition.

Dr. GRAINGER STEWART said the first point which one had to determine was whether the patient was suffering from true trigeminal neuralgia or merely from psychical pain. In the latter class, though operation might cure the patient, the chances were that it would not. He thought it probable that some of the cases which had been reported in which the Gasserian ganglion had been removed, and in which the pain still remained, were probably of a psychical nature. Personally, he had never known the pain to recur after a Gasserian ganglion had been removed. A practical point which, he thought, helped one to realise the true condition of the patient, was that most people who were really suffering from trigeminal neuralgia would not refuse operation, whereas those who could be benefited by medicinal means would do anything to avoid operation. He had not had much experience of alcohol injection. He had tried it in four cases—in old and frail people—and in all the results had been excellent. If the patient was old and the pain could be alleviated for a year or two by the injection of alcohol, such was obviously the line of treatment to adopt; if, on the other hand, the patient was young—and young people did not die of the disease—there was plenty of time to consider more serious measures afterwards. A debt of gratitude was due to Dr. Harris for bringing forward what most of them wanted to know, namely, his results and the difficulties which he had had to encounter, and also for his statement as to the ease with which the operation might be carried out by physicians as well as by surgeons.

Dr. SAMUEL WEST said the discussion had been a very interesting one, though chiefly surgical. He had tried the effect of various drugs for the condition, but the benefit from them was only temporary, and therefore one had been driven in the end to resort to some operative measure. He had not heard nerve-stretching mentioned in the present discussion, he supposed because it was now given up. Still, he had seen some very good results from it, which he thought better than dividing the nerve. Still, matters moved ahead, and we seemed to have arrived at a more distinctly operative stage.

Mr. HUTCHINSON, in reply, expressed his pleasure at having helped to elicit such a valuable discussion, and he thanked Sir Victor Horsley and Mr. Ballance for the kindly way in which they had dealt with his paper, as he, in common with all those present, regarded those gentlemen as masters in neurological surgery. One impression which was made strongly upon him was that the cases must be selected carefully, that some were suitable for peripheral operations, that injections of alcohol, by producing sclerosis and paralysing the nerves, was a good operation, and would take the place of peripheral operations, which had been so largely tried before. But those peripheral operations, right up to the aperture of the skull, had been tried most extensively, and it was only after the repeated failures as time elapsed and as tested by years that surgeons were led to attack the Gasserian ganglion. It was a complete puzzle to him that alcoholic sclerosis at any one point of a nerve should be more successful than such elaborate methods as Professor Thiersch devised, who pulled out whole nerves from close to the skull, or those of Professor Billroth and other surgeons, who devised operations on the second and third divisions. He agreed with Dr. Harris that they must exclude from operation all cases in which a peripheral cause could be found, especially those in which dental irritation was present. But he held most strongly, and he thought Dr. Harris would agree with him, that there were many cases in which there was no peripheral cause. It was certain that the cases which came for the grave operations had mostly had everything tried in the way of discovery of a peripheral cause. He could quote cases where all the healthy teeth on that side had been removed—a wholly useless procedure. He had seen others in which the alveolar margins had been cut away, without any benefit, others in which the antrum had been needlessly opened. He held that there were many cases of true epileptiform neuralgia in which there was no peripheral cause, and there was no central cause ascertainable, though probably there was one present. His own view was that the trouble started in the Gasserian ganglion. He had had many of the ganglia examined, but the most careful research failed to prove pathological change. He did not think there was a neuritis of the ganglion, and no neuritis had been shown to be present in any of the nerves which had been excised in those cases. Some years ago his belief was focussed in the following way: That where those true epileptiform attacks

occurred in two divisions of the nerve, especially in the second and third, it was best to resort to a central operation and not to waste time. Experience proved that there was temporary improvement from a peripheral operation, but there would not be cure; the pain would come back and be worse than it was before, and the disappointment of the patient under those circumstances was very great. He was interested to find that Dr. Harris fully confirmed the exclusion of the ophthalmic in most cases, which justified his belief in that modified resection of the Gasserian ganglion which he had urged as a good operation. With regard to age, he wished to answer the point raised by Dr. Guthrie. The youngest patient in his list was 35, and that was a quite exceptional age. All the others were over 40, the average age of all the patients being 52. So that many were aged 60 or thereabouts; one or two were aged 65. He would be very slow to operate on a young person. One of the most successful cases mentioned by Dr. Harris was where he injected the second and third divisions just at the foramen ovale and the foramen rotundum, and the cure was said to be complete. Was the date of that operation only eight weeks ago? He would not himself form a judgment on a case done only two months ago, it was far too soon. Even exposure of the Gasserian ganglion might cure the neuralgia for many months, but after that it came back worse than ever. Finally, he wished to express his admiration of the physician's skill with the needle. Physicians were encroaching on the surgeons' domain in that line, and he would make them a present of one suggestion: Why not take their courage into both hands and open the skull and inject the Gasserian ganglion with alcohol, and in that way get more lasting cures?

Dr. WILFRED HARRIS, in reply, said he had directed some attention to the point mentioned by Sir Victor Horsley concerning arterial sclerosis. Arterial sclerosis was the one cause which had been put forward to account for the disease. But he had been disappointed. The large majority of his cases could not clinically be shown to have arterial sclerosis. In his patients he had carefully noted the condition of the artery at the wrist, and had taken the patient's blood-pressure, and although in many, as one might expect, there was a blood-pressure of 150 or 160, yet in several the blood-pressure was normal, the artery was felt to be normal, and clinically there seemed no reason to suggest the presence of arterial sclerosis. One case he would like to mention. It was that of a lady, æt. 58 years, who died suddenly of angina pectoris on the very day he was to have injected her with alcohol. If he had injected her the death might have been laid at his door. Her heart was riddled with calcified coronaries, and it was an extreme example of arterial sclerosis. But he had seen several patients who showed clinically no signs whatever of it, and three of his patients he saw, aged forty-two, thirty-five, and thirty-six, in each of whom the neuralgia commenced at the early age of seventeen, showed no special arterial sclerosis. He was a disbeliever in the theory that arterial sclerosis had anything to do with trigeminal neuralgia. Dr. Guthrie had spoken of psychalgia. That, of course, was one of the conditions which had to be distinguished from trigeminal neuralgia, and he thought the Polish Jew mentioned by Dr. Guthrie was a case of psychalgia—a case of mistaken diagnosis before Dr. Guthrie saw him. He (Dr. Harris) had had a similar case sent to him from Bath to St. Mary's for the injection treatment. There was mental pain and crawling sensations all over the head. Obviously it was a case which should not be touched operatively. In the case of tooth-trouble to which Dr. Guthrie referred, her teeth were attended to, but the pain started nine years ago. She had been overhauled by a dentist many times, and he said she was free from tooth trouble. He did not follow Dr. Guthrie when he said the subjects of trigeminal neuralgia were neurotic. He thought it would be agreed by those who had to do with such cases that the majority of them were not neurotic in the slightest degree. His oldest patient started to have the disease at eighty-one, the youngest at seventeen. It had started at all sorts of ages in others, and it was very difficult to know what was the cause. He felt certain there was a definite cause. He believed that the large majority of cases of trigeminal neuralgia were peripheral in origin, and the fact that resection of the affected nerves stopped the pain seemed to prove that in his own mind. If the continuity of the nerve was interrupted at the deep foramen the pain ceased, so long as there was no re-growth of nerve-fibres from the ganglion into the peripheral nerve, which proved, in his opinion, that the pain did not start in the ganglion itself. One advantage of the alcohol injection was that it produced a chemical resection of the nerve, and it left no scar-tissue behind, for the scar-tissue left behind after a surgical operation was a focus of irritation, and might have something to do with the recurrence of the pain. Certainly it was a disadvantage in that it prevented a second operation at the same spot, whereas the alcohol injection could be done many times over at the same spot, for a fortnight after injection one could not find the spot on the skin at which the needle was inserted. With regard to the injection of the ganglion, Mr. Hutchinson had suggested that the physicians should take their courage in both hands and open the skull, injecting the ganglion there by the ordinary route. He had been able many times to inject the ganglion through the foramen ovale on the dead body, but had not as yet attempted it in a living patient.

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CONTENTS.

PAGE

***Three Rare Tumours of the Base of the Tongue, with Remarks on the Surgery of Malignant Disease of the Tongue.** By FREDERIC EVE, F.R.C.S. 113

***Three Clinical Lectures on the Heart.** By SAMUEL WEST, M.D., F.R.C.P. LECTURE II 118

***A Post-Graduate Lecture on So-called Second Sight—its Features, Varieties, and Causes.** By PERCY DUNN, F.R.C.S. 122

* *Specially reported for the Clinical Journal. Revised by the Author.*

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THREE RARE TUMOURS OF THE BASE OF THE TONGUE (TWO OF THEM ENDOTHELIOMATA), WITH REMARKS ON THE SURGERY OF MALIGNANT DISEASE OF THE TONGUE.*

By FREDERIC EVE, F.R.C.S.,
Surgeon to the London Hospital.

THE rarity of endothelioma of the tongue, the uncertain position of these tumours in relation to certain forms of epithelioma, and our almost complete ignorance as regards their course, may justify the publication of these cases.

CASE 1.—Mrs. T. S—, æt. 61 years, was seen by me in July, 1902. She had noticed discomfort in the throat for six months, but complained of no pain or dysphagia. There was a prominent growth on the base of the tongue towards the left side, and some enlarged glands could be felt behind the angle of the jaw.

I operated with Dr. Jekyll, of Leytonstone, shortly afterwards. A preliminary tracheotomy was performed. The external carotid on the same side as the disease was first tied, and the enlarged glands removed. The sub-maxillary triangle was also cleared. The floor of the mouth was opened from below, the tongue brought out, and the right half completely removed. The patient made a good recovery, and is now, at the date of writing, and nearly eight years after the operation, quite well. The specimen preserved in the Royal College of Surgeons Museum (2273 L), showed a smooth, well-defined, prominent tumour springing from the extreme base of the tongue on the left side. It rose abruptly from the surface, its anterior edge projecting three-quarters of an inch at its most prominent part; its diameter was about one

* Delivered before the Clinical Section, Royal Society of Medicine.

and a quarter inches. The surface was not ulcerated, and was covered with smooth mucous membrane. The tumour extended outwards to the pillars of the fauces just below the tonsil, inwards nearly to the middle line, and backwards almost as far as the epiglottis.

A section showed a smooth, somewhat gland-like appearance.

Microscopical appearances.—The epithelium over the surface of the growth was intact, and had no obvious relation with the tumour itself. Beneath the epithelium was the normal adenoid tissue and some channels, evidently dilated lymphatic vessels; a few contained lymph coagula, mostly granular. The growth itself lay deeper down. It was composed of large, irregular, branching and anastomosing columns and masses of cells, and of smaller irregular groups lying in the connective-tissue interspaces. The general appearance of the growth in its outlying parts was that of a carcinoma of the breast. In places the cell-masses showed a definite relation to the channels, presumably lymphatic. They formed bands or cords around them, and occasionally a rounded bud of cells projected outwards from their walls. Strands of cells were also observed in lymphatic channels. Towards the centre of a few cell-masses a well-defined central cavity had been formed by degeneration. Some cells showed colloid metamorphosis, which led to the formation of small, rounded spaces. The cells of the tumour were remarkably uniform. They contained a well-stained, round or oval nucleus with fairly abundant protoplasm, the outline of which was nowhere visible. At no part could a space be seen between individual cells. There were no cell-nests, and no keratinisation was observable.

The growth was an endothelioma associated with dilatation, and possibly over-growth of lymphatics (lymph-endothelioma). The lymphatic glands were not examined with the microscope.

Case 2.—Mrs. H—, æt. 62 years. She had complained of discomfort and a feeling as of a foreign body in the pharynx for two months, but had no pain.

On examination there was a prominent disc-shaped growth occupying the left half of the extreme base of the tongue. Some enlarged glands could be felt in the superior carotid triangle on the same side.

On July 10th, 1909, I operated with Drs. Le Quesne and Anderson, of Tring. A preliminary laryngotomy was performed; the whole tongue was then removed by Syme's, or so-called Kocher's, operation after dividing the mandible in the middle line. Pains were taken to divide the hyo-glossi and genio-hyo-glossi close to the hyoid bone, and especially to divide the base of the tongue as far back as possible. A fortnight later the glands and fat, together with the internal jugular vein, were removed from the whole of the left side of the neck, after dividing the sterno-mastoid muscle. Some of the glands behind the angle of the jaw were one half to five eighths of an inch in diameter, but were not adherent to the surrounding structures.

Convalescence was uneventful, and when I examined her on February 5th, 1910, the patient was quite well and free from recurrence. The specimen now in the Royal College of Surgeons Museum showed a disc-shaped tumour with a well-defined, prominent edge, and a roughened surface covered with fleshy papillæ, but not ulcerated. The growth extended from just behind the circumvallate papillæ backwards to the glosso-epiglottidean folds, inwards to the middle line, and outwards nearly as far as the lateral edge of the tongue.

The section of the growth was pale and homogeneous, its base was ill-defined, and evidently infiltrated the substance of the tongue.

In this tumour also the epithelium formed a thin, even layer over the growth, which extended right up to it. The striking feature of the growth was the presence, especially towards its surface, of numerous large blood-vessels filled with altered blood. Around these the growth formed fairly well-defined columns or cords, separated by thin trabeculæ of connective tissue. In other parts of the tumour the growth occurred in strands or masses, and between these were rounded clumps of adenoid tissue. In the deeper part of the section a few cell-nests were present near the centre of the masses, but they showed no keratinisation. The cells consisted of round or oval well-defined and deeply stained nuclei of an endothelial type. The growth may be classed as a perithelioma. The lymphatic glands were definitely infiltrated. The microscopic appearances closely resembled the primary tumour. There were many large blood-vessels surrounded by cords and columns of cells having well-defined, wavy margins. These

bands also surrounded the connective-tissue trabeculæ, which were thickened. At one spot were whorls of cells, or cell-nests, similar to those which may be seen in an endothelioma of the brain.

Remarks.—These two cases of endothelioma presented many features in common. In each the growth occupied the extreme base of the tongue and formed a very prominent disc-shaped mass which was not ulcerated on the surface. Glands were distinctly enlarged in the superior carotid triangle in both. The symptoms had been discomfort or a feeling of a foreign body in the fauces, and were so slight that the growth attained a considerable size before it was discovered. The microscopic character of the tumour in the first case was of a lymph-endotheliomatous type, the growths consisting of cord-like masses of endothelial cells, in places surrounding dilated lymphatic channels.

In the second case it was peritheliomatous. There were very numerous and large vascular channels surrounded by cords of endothelial cells.

Notwithstanding the early and marked enlargement of glands, the fact that the first patient has survived for nearly eight years leads one to hope that free and early removal of this disease may be followed by satisfactory results. But it should be pointed out that the outline of this tumour was unusually well defined. I may say that the endotheliomatous nature of this tumour was apparent to me when I removed it in 1902, and I sent it to the Royal College of Surgeons for preservation.

The occurrence of endothelioma in the base of the tongue can be readily understood. This portion of the organ is made up of a considerable amount of adenoid and lymphatic tissue, and has been called the lingual tonsil; it also contains many mucous glands, and in this respect resembles the salivary glands, in which endothelioma is the commonest form of growth.

In the 'Archives of Middlesex Hospital,' vol iii, 1904, Dr. Lazarus Barlow has reported four cases described as examples of endothelioma of the tongue. They were met with in 105 cases of malignant disease of the tongue, between the years 1900–1903, both inclusive.

All the patients were males, their ages being respectively 42, 44, 51, and 56. In two instances the disease occurred at the base of the tongue, and laterally as in my own cases; and in the other two the growth was close to the tip. Deep erosions of the superficial portions of the growth characterised all the cases. In this respect, therefore, Dr. Lazarus Barlow's cases differed materially from those described above. In every instance there was enlargement of the infra-maxillary glands. The disease ran a rapid course in three cases, and the second case was lost sight of. The first case lasted one year from commencement to death, the third case two years, and the fourth case fourteen months. At the post-mortem examination in Case 1 an extensive growth was found in the mandible, together with growths in the submaxillary glands and liver; in Case 3 there were nodules in the submaxillary glands and lungs, and in Case 4 growths in the submaxillary glands and liver. In two cases the growth was of a lymphangiomatous type, and in the other two it was peritheliomatous.

Case 3.—*Epithelioma without keratinisation.* Mr. B—, æt. 52 years. He had noticed soreness and a swelling at the base of the tongue three months before he consulted me on July 21st, 1909. There was a prominent growth projecting from the extreme base of the tongue on the right side. Its surface was slightly papillary but not ulcerated. Its edges, abruptly raised a quarter to three eighths of an inch above the base of the tongue, were overhanging. The growth sprang from the outer two thirds of the extreme base and extended outwards as far as the tonsil, and backwards to the glosso-epiglottidean folds. In fact the extreme posterior edge passed downwards along the pharynx to a level behind the anterior surface of the epiglottis.

On July 24th, 1909, I operated with Mr. Mark Hovell. The tongue was removed by Syme's method, after a preliminary laryngotomy. I also removed the pillars of the fauces together with the tonsil; and although posteriorly I got behind the limit of the growth, I did not feel that I had been able to remove a sufficient margin of healthy tissue. On August 9th the whole of the right side of the neck was cleared of glands and fat, the internal jugular vein being tied below and stripped upwards with the glands. Many of the glands in the superior carotid triangle were very large, but were not adherent. The sterno-mastoid muscle was divided. Six months later I examined the patient, and found a small, raised, non-ulcerating

growth on the scar in front and to the right of the epiglottis, and extending to the antero-lateral wall of the pharynx. The margin of the right half of the epiglottis itself was also distinctly thickened. After consultation with Mr. Butlin and Mr. Hovell it was decided to remove the larynx together with the recurrent growth. I performed this operation on February 10th. The incisions were confined to the middle line and left side of the neck, as the skin on the right side of the neck was a good deal thinned by scarring, and by the free removal of tissue at the previous operation. The left external carotid was first tied; then the trachea was divided below the cricoid, without a preliminary tracheotomy, and the larynx was separated by dissection from below upwards. The next step was to divide the hyoid bone in the middle line, turn it upwards with its attached muscles, and then open the pharynx freely. The recurrent growth was widely removed in one piece with the larynx. A nasal tube was kept in for about ten days. The patient left for home quite well three weeks after the operation.

The primary growth in the tongue was made up of anastomosing columns and strands of cells supported by narrow connective-tissue trabeculæ. The cells contained large nuclei, and the outline of their protoplasm was in parts well defined. Several whorls of cells were observed, but there was no keratinisation. Many of the cells were vesicular. There was no alteration of the blood or lymphatic vessels. The glands were not examined. At first I thought that this growth was also an endothelioma, but the well-defined outline of the protoplasm of the cells and the fact that many of them were vesicular led me to have fresh sections prepared, which included the surface of the growth. It then became apparent that the columns of cells extended down from the surface in the intervals between large papillæ; the epithelial covering of these had, however, broken away in the process of cutting the sections. The tumour was, therefore, an epithelioma springing from the cells of the Malpighian layer, and whose component cells remained undifferentiated. It can readily be understood why this form of growth occurs at the base of the tongue where the epithelium is not keratinised, and is practically similar to that of a mucous membrane.

I have included this case for various reasons. It appears to me a rare form of malignant disease of the tongue, and unless careful discrimination is made, might readily be mistaken for an endothelioma. Further, its position at the base of the tongue, its abrupt steep margins, and the absence of ulceration render its naked-eye characters very similar to the cases of endothelioma I have described above.

In Butlin and Spencer's excellent work on 'Diseases of the Tongue' (p. 313), an apparently similar case is quoted and figured (fig. 29). It occurred in the practice of Mr. Spencer. The patient, a girl, æt. 22 years, had observed an ulcer opposite the left lower molar teeth for two months. In two and a half months from its first appearance the growth had involved half the tongue throughout its whole length, the floor of the mouth, the surface of the tonsil, and the adjacent portion of the soft palate. In spite of early operation she died within ten months of the commencement of the disease. The growth consisted of masses and columns of cells infiltrating the muscular fibre, without forming cell-nests.

I may say that I have to thank Mr. Shattock for very kindly studying the microscopic sections from these cases with me; and he concurs in my description of the appearances.

Remarks.—A few remarks may be made on the surgical aspects of the cases. In the first case half the tongue was removed through a lateral incision in the neck after ligaturing the external carotid and removing the lymphatic glands. I had not at that period (1902) adopted Syme's or Kocher's operation of dividing the mandible in the middle line. The obvious disadvantage of removal of the tongue from below is that it leaves a large wound in the neck which must necessarily become septic as it communicates with the mouth. And when the whole tongue requires removal I have not found it easy to expose the opposite base.

In the second and third cases the tongue was excised by severing the mandible, and the glands were removed at a second operation. This I consider by far the best method of removing any growth in the tongue of considerable size, especially if it extends as far back as the anterior pillars of the fauces; and still more so if it involves the floor of the mouth. For, contrary to a high authority, I do not consider that any growth implicating the floor of the mouth can be satisfactorily removed without dividing the mandible. The great advantages of Syme's operation are: That the lingual

arteries are easily secured, far back, after division of the hyo-glossi; the tissues of the floor of the mouth can be cleanly detached from the mandible; and the genio-hyo-glossi, hyo-glossi, etc., together with the tongue itself, can be severed at their attachments to the hyoid bone. Whatever may have been written to the contrary, I consider that, exclusive of quite small growths, the muscles of the tongue should be divided near their several origins, due regard being paid to the situation of the growth and the direction of its extension. When the growth is ulcerated, and especially if foul, it seems to me important that the tongue should be excised first. For it appears likely that a wound in the neck may be infected with septic matter from the lymphatics of the tongue; and it is possible that the wound may also be inoculated with carcinoma. In any case the patient's general condition is improved by the removal of a septic mass from the mouth.

The disadvantages of Syme's procedure are in connection with the union of the two halves of the mandible. On four occasions I have had to remove the wire which united them owing to the existence of a sinus; and such a sinus may necessitate inconvenient delay in the performance of the second operation on the neck. Delayed union has also occurred in my practice owing to the absorption of bone around the wires, and loosening of the fragments. This may also lead to irregularity of the teeth. These disadvantages can be entirely avoided by having a dental splint prepared beforehand, which can be fitted at the end of the operation.

The third case related is an example of a growth extending very far back along the anterior wall of the pharynx to a level posterior to the anterior surface of the epiglottis. It also passed upwards on the lateral aspect of the fauces and involved the tonsil. I removed it by Syme's method, but found great difficulty in getting sufficiently far behind it, and recurrence occurred in the pharynx and epiglottis after eight months. In this case I think it would have been preferable to perform the operation on the affected side of the neck first, removing the glands and tying the branches of the external carotid by Dawbarn's method. Later, the whole tongue might have been removed by incisions limited to the sound side of the neck, after a method recently published by Mr. Butlin for such cases.* This procedure was adopted for the removal of the recurrent growth, but since the epiglottis was involved, it was necessary to combine with it laryngectomy. An illustration is thus afforded of the usefulness of this method for extensive growths implicating the base of the tongue, fauces, and pharynx, because if it be found after opening the pharynx that the larynx is affected, there is no difficulty in modifying the operation originally planned so as to include a laryngectomy with excision of the tongue. A partial removal of the larynx is probably useless. Butlin has found that if the disease is attacked from the affected side of the neck (viz. that on which the anterior triangle was cleared), a hole communicating with the pharynx may be left, which retards the patient's recovery for many weeks (*op. cit.*, p. 225).

Regarding the removal of the glands of the neck, it is scarcely necessary to say that not only the glands, but the fat and loose connective tissue should be removed from the affected areas. For some years, in this and similar operations, I have stripped up the fat and connective tissue from the surface of the muscles with gauze pads. The method is expeditious, makes a clean dissection, and is attended with little hæmorrhage.

It is sometimes necessary to clear the whole side of the neck, for it may be found that enlarged glands extend under the sterno-mastoid into the posterior triangle, especially in its upper half. The usual incision is made from the mastoid process to the clavicle, but, instead of carrying it along the anterior border of the muscle, I carry it over its middle, so that the whole width of the muscle and the surface behind it can be readily exposed. A second incision is made from the centre of the vertical one to the chin, and the flaps outlined are turned upwards and downwards respectively.

In most cases I divide the sterno-mastoid just below the exit of the spinal accessory nerve, so that the halves of the muscle can be turned downwards and upwards without hindrance. The internal jugular is tied low down, divided, and the connective tissue cut through just above the clavicle. The internal jugular, glands, and fat, are then dissected up by gauze stripping, a look-out being kept for the thoracic duct on the left side. I have found it convenient to clear the submaxillary triangle from above. After ligaturing

* 'Operative Surgery,' edited by F. F. Burghard, p. 224.

the facial artery and vein, where they lie on the jaw, a horizontal incision is made through the connective tissue just below the horizontal ramus. The lymphatic glands, fat, and submaxillary salivary gland are then stripped downwards and backwards. Wharton's duct is tied before division, as I think it may infect the wound from the mouth. After dissecting downwards and backwards the contents of the submaxillary triangle with gauze and scissors, only the facial artery at its origin and the common facial vein remain to be tied. Finally the dissection of the internal jugular upwards to the base of the skull is completed, and the proximal end of the vein is secured. In those cases in which glands in the posterior triangle are extensively involved, I make an incision from the centre of the first vertical incision, backwards and obliquely upwards towards the spine of the axis, and expose the posterior triangle by reflecting the flaps marked out by it.

I am in agreement with those who think that, as a rule, it is only necessary to remove the glands on the affected side unless those on the opposite side are actually enlarged. The exceptions to this rule are, however, numerous. They include all growths in the tongue originating in or approaching the middle line, those deeply infiltrating the under surface of the tongue, those arising in the tip of the tongue, or implicating the floor of the mouth at or near the frænum.

May 30th, 1910.

Importance of Abdominal Breathing in Case of Weakness and Insufficiency of the Heart.—Rumpf has been teaching for years that greater attention should be paid to regulation of breathing with heart disease. The art of breathing properly should be emphasised; especially in heart disease costal breathing should be suppressed as much as possible and abdominal breathing promoted. Röntgen-ray examinations show that with abdominal breathing the lungs follow the diaphragm downward, with traction on the apex of the heart, pulling it more toward the vertical line. This checks the tendency to dilatation. The Röntgen rays show how the left outline of the heart is drawn inward and down, thus making the left heart smaller, and in some cases also the right. —*Med. Record*, vol. lxxvii, No. 20.

THREE CLINICAL LECTURES ON THE HEART.

Delivered at St. Bartholomew's Hospital.

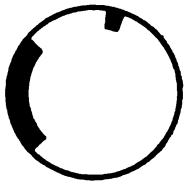
By SAMUEL WEST, M.D., F.R.C.P.,
Physician to the Hospital.

Lecture II.

Gentlemen,—In my last clinical lecture I dealt with various points connected with the heart, and I turn to-day to the subject of murmurs. We must understand first what is meant by a murmur; I give you a diagram of the heart-sounds which ought to make it clear. If two things are heard distinctly it is obvious that they must be separated by an interval, otherwise they would run into one another and would not be heard distinctly; so the heart-sounds must be separated by distinct intervals. During those intervals in a healthy heart we hear nothing, but in diseased hearts we may hear something, and that something is a murmur. So a murmur may be defined to be an abnormal noise heard between the two heart-sounds. This makes clear the essential distinction which must be drawn between murmurs and heart-sounds. Heart-sounds may be present with murmurs or they may be absent, or they may be altered in various ways. To speak of murmurs replacing heart-sounds, as is commonly done, is inaccurate. There is no doubt whatever about this in the case of the second sound, which is too short for a murmur to replace it and do nothing more. The appended diagram may be taken as representing the cardiac cycle. It differs from that found in many books. I have drawn the first sound as not occupying more than half the systolic period, whereas it is often made to occupy the whole, fading gradually away until the second is heard. Such a diagram does not represent what the ordinary ear hears, or what, in my opinion, any ear can hear. Anyway, there are many cases in which a systolic murmur is heard and yet the two sounds appear perfectly normal in every respect, while the murmur appears to be as long as the first sound—that is, the two divide the systole equally between them, and in this case, at any rate, the first sound cannot be drawn as lasting beyond the middle of systole. Of course it is impossible to say what another can, or cannot, hear. Some

persons cannot hear the grasshoppers on the Swiss mountains, while to another their noise is almost painful. As there was for a long time no means of actually measuring it, the length of the first sound remained a matter of opinion. Recently, by an ingenious arrangement, actual tracings of the heart-sounds have been obtained,* which show that the diagram as I have drawn it is correct. Indeed, I cannot see how careful clinical observation can lead to any other conclusion. If the drawing of the first sound given in so many of the physiological text-books be accepted, no distinction could be made between the first heart-sound and a murmur, and a systolic murmur could not be distinguished from an altered first sound. Yet at the bed-side it can and must be. It may be of no great importance physiologically whether the diagram be right or wrong, but it is very important in medicine, for upon it depends the correct definition of a murmur.

How is a murmur produced? It is due to what French writers have called a fluid vein, or,



in other words, a jet of blood, which is driven with some force into the blood-stream. This jet causes eddies, and those eddies cause noises, and those noises are murmurs. Now, the extraordinary thing about the heart is not that murmurs occur in disease, but that they do not occur in health. So beautifully adjusted are the vessels and the heart to one another that the blood passes in and out without eddies or currents, for all eddies and currents mean loss of power and friction. Yet it is not to be wondered at that even very slight disturbances of the relation between the vessels and the cavities of the heart in disease should cause the occurrence of these eddies upon which murmurs depend. And that is what happens.

There are two ways in which a disturbance of these relations can be produced—either by alteration in the orifice itself or by dilatation above or below it. In either case eddies will arise and murmurs be produced.

Murmurs are classified according as they are associated with some organic lesion in the heart or not. Those which are not associated with organic disease are sometimes called "functional" or "hæmic murmurs," but neither of these terms is free from objection. To determine at the bedside whether a murmur is organic or functional is often a difficult problem, for the solution of which great care and experience may be required. Among the organic murmurs there are two groups again, according as the lesion involves the valves, when the murmurs are called "valvular," or not. The valves are the aortic, pulmonary, tricuspid, mitral, each of which may be diseased, and at each of them organic murmurs may be produced, the murmurs being named accordingly.

If a murmur be heard the first thing to decide is whether that murmur is systolic or diastolic. Here a difficulty arises which has been the cause of much confusion. The first sound is sometimes spoken of as the systolic sound, because it marks the commencement of systole, and the second sound the diastolic, because it marks the commencement of diastole; but systole and diastole are periods of time of some duration. The adjectives systolic and diastolic should refer to these periods, but they are sometimes used as if applying to the sounds. A systolic murmur, therefore, should be one which occurs during systole, and a diastolic murmur one which occurs during diastole. In the case of a systolic murmur, if the first sound lasts, as it is drawn in many books, right through the whole of the systolic period, it would matter little whether systolic referred to the sound or the period; but if, as I have stated, the first sound does not last more than half way through this period, then it matters much. For instance when we speak of a post-systolic murmur, if systolic applies to the first sound, post-systolic means after the first sound and therefore before the second sound. And therefore the post-systolic murmur would come in what is the systolic period, and that is an absurdity, for it cannot be systolic and post-systolic at the same time. Diastolic or post-diastolic present the same difficulty.

But a further limitation of the terms is necessary still, for all parts of the heart do not contract at the same time. The two auricles do and the two ventricles do, but the auricle contracts just before the ventricle, and therefore the auricular systole occurs during the last part of the ventricular

* Einthoren and Geluk, 'Arch. f. d. Ges. Physiol., 1904, lvii, 607.

diastole. When we speak, then, of systole and diastole we refer to what the ventricle is doing, unless we put the prefix auricular before it and speak of the auricular systole and the auricular diastole. A systolic murmur, therefore, means a murmur which occurs during the ventricular contraction, and a diastolic murmur one which occurs during the ventricular diastole.

To time the murmur, *i. e.* to find out whether the murmur is systolic or diastolic, is easy enough by feeling the pulse; the heart's impulse is not convenient, and the pulse at the wrist is not accurate enough, but the pulse in the carotid is practically synchronous with the ventricular contraction. If, therefore, the murmur heard comes with the pulse-beat in the carotid, it occurs during the ventricular contraction and is a systolic murmur. If it does not come with the pulse it is not systolic and must therefore be diastolic. It should be the rule never to auscult a heart for the first time without having the finger or thumb upon the carotid, for systole and diastole cannot always be determined by the ear alone.

As regards the characters of murmurs, they may be long or short, blowing and soft, or harsh and noisy, and sometimes musical. These terms speak for themselves. Blowing murmurs often do not depend upon organic disease, but harsh ones almost invariably do. The murmurs, whether systolic or diastolic, may vary in intensity from one day to another, or even at short intervals, *e. g.* while you are listening to them. This generally depends upon the force with which the heart is beating. When a jet is produced by the action of the heart, if the action be very slow and feeble the jet will not be a forcible one, and the noise or murmur weak. If the action be strong the jet will be forcible and the noise loud. When a patient comes into hospital exhausted and ill, the murmurs may be indistinct. If you are wise you will not attempt to make a diagnosis then but will wait a few hours, and then you may find that the murmurs are distinct, and what was at first obscure has become clear. Very often patients who come into the hospital with a slight murmur develop a loud one before they leave. It does not necessarily follow that the disease has made great progress and that the patient leaves the hospital worse than when he came in. It may mean, on the contrary, that he is very much better, and that the heart is beating strongly at the later date, whereas previously it was acting weakly. Again, when the patient is getting worse the murmurs which were at first distinct may become weaker and weaker, and finally inaudible.

Murmurs may also vary with the position of the patient. They may be distinct when the patient is sitting or standing and feeble or absent when lying down, or *vice-versâ*. If, then, you have a murmur which you do not understand, change the posture of the patient and see what effect is produced; often this will make what was doubtful clear.

Having decided that there is a murmur, and that it is systolic or diastolic, as the case may be, we now want to know what is the source of it. Where does it originate? Murmurs are divided as I have said into two classes—those that are valvular, *i. e.* due to disease of the valves, and those that are not. We will consider the valvular ones first.

The first thing we have to observe is the direction of propagation. Murmurs are propagated in the direction which that jet of blood takes which causes them. They are different, of course, at the different orifices of the heart.

Aortic murmurs.—Take the aortic orifice first. If I close my left hand, and place it in the lower part of the sternum, it is about the size of my heart; if I straighten out my thumb it points up to my right sterno-clavicular joint. I might draw a line from this point to the apex, and thus describe what I have named the "aortic axis." It is along this line, up and down, that aortic valvular murmurs are propagated. If the jet of blood producing the murmur be driven upwards into the aorta when the heart contracts, the murmur will be propagated upwards along that line to the right sterno-clavicular joint and into the vessels of the neck. If the jet passes the other way when the heart is dilating the murmur is propagated downwards towards the apex, and very often these murmurs can be heard quite distinctly down that line even to the apex. At its origin the aorta is covered in front by the pulmonary artery. As these vessels cross and diverge the aorta becomes clear of the pulmonary in the right second space and this is called the aortic area. The place where you can get nearest of all to the root of the aorta and be free of the pulmonary artery is about the third left intercostal space. This is a

very important fact, because it is often in that particular spot that diastolic aortic murmurs are heard loudest, or it may be there only and nowhere else. The third left intercostal space then near the sternum is the most important place to listen for aortic diastolic murmurs, just as the second right space is for systolic murmurs. Sometimes the diastolic murmurs are propagated louder down the sternum, and this has been referred to diseases of the anterior cusps of the aortic valves.

The next thing about these murmurs is to bear in mind that the loudest murmur does not necessarily mean the most extensive disease. If when the three cusps of the aortic valve meet there is a tiny hole left or if one of the valves has a perforation in it, the amount of aortic disease would be very slight and might be even overlooked at the autopsy, yet there might have been a very loud murmur during life, and that, too, a musical one. Why? Because there is a good big, healthy ventricle, which pumps blood with considerable force and fills the aorta well, so that when the valve is closed there is a good deal of tension in the aorta and the blood passes back with considerable force through that narrow little opening. So the noisiest murmurs do not necessarily mean the largest amount of organic mischief. At the same time the opposite is also true, that with an enormous amount of disease the murmurs may be soft and faint, and there may be little more than this when there are no valves at all. So that we cannot conclude because a murmur is soft that there is no great amount of disease, or because it is loud that there is a large amount of disease. It is easy enough when you have discovered murmurs which are propagated along the aortic axis, to conclude that the patient has aortic disease.

The next thing to consider is the effect which the disease would have on the heart. If the left ventricle has to pump the same amount of blood through a narrowed orifice it wants more power to do it, and will therefore hypertrophy. Again, if the valves are incompetent, so that the blood passes back from the aorta during diastole, the ventricle will be dilated, or both dilated and hypertrophied. So the apex of the heart, instead of being in the fifth space, about an inch inside the left nipple line, may be in the sixth space, a little under or outside the left nipple line, and the cardiac dulness will follow the apex—it may be still triangular, but it will be longer and larger.

The character of the radial pulse will be also changed. If the aortic orifice be narrowed so that the blood cannot pass readily through it, the pulse will be altered in one way. If, on the other hand, the aortic orifice be so diseased that the valves do not close properly the pulse will be altered in another way. There are pulses which are characteristic of aortic stenosis on the one side and of aortic incompetence or regurgitation on the other. There are two forms of aortic stenosis; in the one the thickening is most marked at the attachment of the valves to the aorta and in the other at its margins. In either cases the valves may meet, so that there is no incompetence. There will be obstruction in both, so that the blood will find a difficulty in passing through the orifice.

A healthy pulse-tracing shows a sudden upstroke, a short wave, and a gradual fall. The upstroke is due to the sudden driving of blood into the vessels. With stenosis of the valves the blood cannot be driven suddenly into the aorta, though it trickles through in time, consequently the upstroke will be low, the wave long, and the pulse-rate slow. Aortic stenosis pure and simple, however, is the rarest of the valvular affections. On the other hand, aortic regurgitation is common, and alters the pulse in a very marked way. This characteristic pulse has received special names—Corrigan's pulse, the water-hammer pulse, the pulse of emptied arteries—but it is best called the pulse of aortic regurgitation. The upstroke is high, the drop sudden, and the wave very short. The upstroke is high because the left ventricle is hypertrophied, the drop is abrupt because there are no valves to keep the tension up, and consequently also the wave is short. This is the typical pulse, but even with marked aortic regurgitation we do not always get it. Let us consider why. In the first place the valves, which are so much diseased as to be incompetent, may at the same time be so much thickened as to cause much obstruction too. Then the typical forcible upstroke will fail. There is another reason still, for the ventricle may be too feeble to contract forcibly at all, and so we may see a well-marked Corrigan's pulse lose its typical characters as the left ventricle becomes more and more feeble.

Now let us sum up what we have got as regards aortic disease. Murmurs are audible, systolic or diastolic as the case may be, and they are propagated in certain directions. They are attended with certain changes in the heart and certain alterations in the pulse. When we have all these, or most of them, we can diagnose with certainty that the seat of lesion is to be found at the aortic orifice, and we diagnose the case as one of aortic disease

Mitral murmurs.—Let us now turn to the other set of valves, the mitral. How is mitral disease to be diagnosed? The murmur heard is systolic, or diastolic as the case may be, and now we want to know where it is heard loudest, and in what direction it is propagated. Mitral murmurs are heard at the apex. Is there anything which you can speak of as an axis in mitral disease? If you were to take a long skewer and put it in at the position of the normal apex and bring it out behind in the left interscapular space a little above the angle of the scapula, you might theoretically pass through the mitral orifice; this skewer may be taken to represent the mitral axis. The two points where this axis comes to the surface are (1) the apex in front and (2) above and within the angle of the scapula behind.

The left auricle is behind and the left ventricle in front, so that the blood in passing from the auricle to the ventricle in diastole passes from back to front. If on systole a jet of blood passes through the valves, *i. e.* if the valves are incompetent, it will be passing the other way from the front to the back. A systolic murmur heard at the apex in front and in the left interscapular space behind will be due to a jet of blood passing through the mitral orifice backwards, and the conclusion is that the mitral valve must be incompetent. Or to put it the other way, the murmur of mitral regurgitation is heard at the apex in front, and near the angle of the left scapula behind. The matter is not quite as simple as that, because these murmurs are not propagated in those directions only, but are sometimes rather widely heard in the axilla as well. But the broad facts stated are sufficient for our present purposes. On the other hand, if the murmur be diastolic and produced when the blood is passing in its proper direction from the auricle into the ventricle, *i. e.* from behind forwards, the murmur will be heard in front, but not in the left intra-scapular space behind. Consequently if we have a systolic murmur heard at the apex and behind we conclude the patient has incompetence of the mitral valve; while if there is a diastolic murmur heard at the apex in front and not behind we diagnose that there is mitral narrowing or stenosis.

The apex-systolic murmurs we do not classify; they are simply long and short. When long the increase of length is at the expense of the first sound, which may even be absent entirely, so that the murmur then occupies the whole of systole. The diastolic period is longer and may be divided into three parts, the murmurs being accordingly named early, middle, or late diastolic. The late diastolic, occurring as it does just before the systole, is generally called a pre-systolic murmur; this is the characteristic murmur of mitral stenosis. Just before the systole of the ventricle the auricle contracts, so that the pre-systolic murmur has been also called the auricular systolic—a good enough name, but one which has gone out of use and will not, I hope, come into use again, for clearness is gained if the terms systole and systolic, diastole and diastolic, be restricted to the action of the ventricle only. If this be done and it be remembered that systole and diastole are periods of time, and that systolic and diastolic refer to these periods and not to the heart-sounds, there will be no confusion about murmurs.

Tricuspid and pulmonary murmur.—With regard to the two other valves, the tricuspid and the pulmonary, it is necessary to say something about the direction in which the murmurs produced at their orifices are propagated.

The axis of the pulmonary artery runs from the third right costo-sternal articulation to the middle of the left clavicle, and it is along this line that pulmonary murmurs are propagated. A pulmonary diastolic murmur is the rarest of all the murmurs.

The tricuspid valve lies close beneath the sternum, and the axis may be represented by a horizontal line running across the sternum to the right at the level of the fifth costo-sternal articulation, and it is along this line that tricuspid murmurs are propagated. You will see the importance of this when we speak of the murmurs of dilatation, as I propose to do in a subsequent lecture.

May 30th, 1910.

A POST-GRADUATE LECTURE

ON

SO-CALLED SECOND SIGHT—ITS FEATURES, VARIETIES, AND CAUSES.*

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In the popular world it is not infrequent to hear of the recovery of vision in old age. The story generally runs: "Isn't it wonderful? Mr. X—, who is over eighty, is now quite able to read without glasses." And there is always just the suspicion of the story conveying the impression that somebody was at fault in ordering glasses at all. Possibly, too, the old patient himself feels some satisfaction

* West London Post-Graduate College.

in being able to say to his ophthalmic surgeon, "Those glasses which you ordered me last are unnecessary. I can see to read quite well without them." The belief, therefore, gains currency that some marvellous change has occurred, and that the power to read without glasses at such an age is merely confirmatory of the remarkable vitality which the old person has hitherto exhibited. Of course the popular world knows nothing of the causes underlying this apparently miraculous reacquisition of sight, and it is regarded as being miraculous mainly because the possibility of its occurrence had never been foreshadowed. Nor is it possible for anyone to forecast a result of this nature. The conditions favourable to its incidence may be present in particular cases, but in no case, with any certainty, can it be said that this apparently miraculous achievement will ensue. Nevertheless, if any compensations do exist which can be placed to the credit of old age, this is one, perhaps, which, so long as it lasts, might be included.

The condition of so-called "second sight" is by no means the simple matter which its name would tend to imply. On the contrary it is based upon many interesting details which belong to the ophthalmic surgeon's work. It even involves points of pathological obscurity, the solution of which is still undetermined; moreover it constitutes, perhaps, the only instance in the body in which a degenerative process is associated with advantage to the individual.

Let us, however, clear the ground somewhat of our subject by taking a typical case of the kind. A gentleman, who is emmetropic, at forty-five requires correction for his presbyopia. This correction is continued, with the necessary change in the focal length of the lenses, until, say, about the age of seventy is reached. Then complaint is made that the distant vision is not so clear as formerly. Distant objects are not so distinct as they used to be, nevertheless some improvement in the distant vision is obtained by means of low concave glasses. Then it is found that the convex lenses in use for reading are not satisfactory, that weaker ones than those hitherto worn give better results, and at last it comes to pass that fine print is seen best without any glasses at all. Thus the patient has attained to that condition, known as "second sight," as the result of which he becomes the object of wondrous comment among his friends, and is glad enough to display, whenever he can, the power which he has thus regained.

And yet, as a matter of fact, the change which has brought about this apparently miraculous improvement in the near vision is not in any sense miraculous. On the contrary, it is due to a degenerative process concomitant with advanced age; it is neither indicative of a special vitality, nor is it proof of a condition which justifies the friendly out-pouring of so much congratulatory comment.

In inquiring as to the locality of the degenerative process in question, it is essential to recall that the first ocular structure to indicate the progress of age is the lens. With the advance of age this structure exhibits a gradual decrease in its elasticity, due in part to its condensation, depending upon the loss of water, which tends to produce a process of sclerosis, the effects of which are first apparent in the nucleus. Thus with the hardening of the lens its elasticity becomes more and more impaired, and despite the relaxation of the zonule accomplished by the ciliary muscle, the lens ultimately ceases to be able to change its shape. It is owing to these lental changes that presbyopia supervenes.

This failure of nutrition, however, in the lental body, while correctly regarded, perhaps, as a physiological process, may subsequently emerge into one which is distinctly pathological. When this is the case the lens then begins to exhibit changes which indicate the development of cataract, and the difference between the physiological and pathological changes in the lens in this regard is, that while the former only causes loss of accommodation described as presbyopia, leaving the distant vision in an otherwise emmetropic eye unchanged, the latter renders both the distant and near vision defective. But it may be asked, in what way is this difference concerned with the advent of so-called "second sight"? In reply to this question we must revert to what is known as the "pathology" of cataract. I use advisedly the expression, "what is known," since, possibly to the surprise of those unfamiliar with the subject, it is a fact that the pathology of cataract is still a matter of mere speculation, and occupies a nebulous position so far as accurate knowledge is concerned. Nevertheless, the lens has been the subject of much interesting inquiry respecting its normal

nutrition and pathology. Indeed, a study of its literature reveals that an immense amount of original work has been devoted to the elucidation of these special points; and while it is true that original investigation has established some accuracy of knowledge regarding the processes of nutrition of the lens, and the changes which ensue during the progress of cataract, the problem nevertheless still remains unsolved in respect to the real ætiology of the disease. Naturally enough many theories have been advanced to supply the place of knowledge thus wanting. I do not, however, here intend to discuss the various suggestions which have been made to account for the occurrence of senile cataract; that it is a nutritional change dependent upon defective systemic metabolism seems to be proved by its incidence in association with diabetes. Metchnikoff, in his recent very suggestive and interesting work on the 'Prolongation of Life,' alludes to the development of cataract in old age. His general theory is that the failure of nutrition in old age "is due to the action of devouring cells," which he calls "macrophags"—in short, phagocytes—"which destroy the higher elements of the body, such as the nervous and muscular cells, and the cells of the liver and kidneys." He, however, admits that the eye is an organ that is modified in old age without the action of macrophags, for the reason, as he states, that the cornea and the lens contain no cells out of which macrophags could be formed. It must be confessed that this admission, based upon scientific fact, seems rather to discount the general theory which he has evolved. If his law of the failure of nutrition in old age be true of one tissue, it should be true of all tissues of the body in which the atrophy of senescence is observed. Arcus senilis, so-called, of the cornea is due to the deposition of fine granules of fat in Bowman's membrane, and in the lamellæ of the cornea; and in cataract the opacity is due to an accumulation of a fatty substance in the lens-fibres. But, says Metchnikoff, "in most organs such fatty degeneration is followed by phagocytosis, a result, however, from which, for anatomical reasons, the cornea and lens are exempt." Thus, although Metchnikoff's views cannot be said to throw any light upon the ætiology of cataract, yet I especially allude to them, inasmuch as no reference to the results of the failure of nutrition in old age would in these days be complete without taking into consideration the attractive, original, and interesting suggestions which he has made upon the subject.

It is not, however, with the ætiology of senile cataract that our subject of "second sight" so called is so much concerned, but rather with the pathological changes in the lens associated with the development of the disease. Here we stand on firmer ground from the point of view of accuracy of knowledge. The changes which a cataractous lens undergoes in the progress toward maturation have been definitely and clearly determined. It is, therefore, necessary to consider these somewhat in detail, since it is the incipient pathological changes which define the onset of cataract to which the condition of one variety of so-called second sight is due. Thus, contrary to the popular belief, this condition is not one in any way indicative of a "marvellous" vitality; its occurrence implies a degenerative process which tends to affect senile tissues generally.

The researches of Becker have shown that the loss of transparency in the lens, in the incipient stage of senile cataract, is due to commencing separation of its fibres. This separation is supposed to depend upon the shrinkage of the sclerosed nucleus, the result being that clefts or spaces are formed between the fibres, in which fluid collects. Owing to the imbibition of fluid the lens swells, by which its refractive index is increased. Objectively this change in the size of the lens is indicated by a narrowing of the anterior chamber, and the increase in the lental curvature is shown by the development, in some degree, of myopia. It is this lental myopia which is concerned in the initiation of those changes which account for the phenomenon of this form of second sight. With the development of the myopic condition there ensues a diminution in the acuity of distant vision, which loss of sight is also partly due to the commencing decline in the transparency of the lens. Thus a patient is unable to see distant objects as clearly as before, while the effect of the acquired myopia, according to its degree, is to neutralise, wholly or in part, the presbyopia which has been present. Thus, while the distant vision is improved by the use of concave lenses, on the other hand the near vision may be found to be best without any glasses at all. The interest in these cases really lies in the course which the cataractous process takes.

If it proceeds onwards to maturation the vision fails generally, *pari passu*, with the advance in the lental opacity, for which, of course, there is no optical remedy. Occasionally, however, it happens that in old persons the process advances so imperceptibly that useful vision is retained until the end of life. In such cases the old patients ignore the failing vision for distance, and are only too pleased that the near vision remains useful for reading without glasses. The older the patient is when cataract begins the slower and less complete is its advancement. This chronicity of a pathological process in old age is familiar to us in connection with other diseases. Scirrhus of the breast, for example, in an old woman may be of so non-malignant a type that the patient may ultimately die of some intercurrent disease without the cancer having shortened her life. Thus it is that we can never predict with any certainty how a cataract will progress. In every case its progress varies. Even when occurring in middle life it sometimes advances so gradually that useful vision is for some years retained. A patient, therefore, in whom there are signs of incipient cataract should never be informed of the condition present. For various reasons it is better that the fact should not be divulged, except in confidence, to the immediate friends concerned. I may mention two instances in which a premature statement in this regard was resented. A lady informed me one day that she had been grossly insulted by an ophthalmic surgeon telling her that she had cataract. It was true that faint striæ were visible in her lenses. But she was only forty-five years of age, and from her point of view to be candidly informed that she was suffering from a disease from which she believed that only old people suffered was insulting and intolerable. Thus, from this aspect, it is plain that the premature announcement to a patient that he or she is the subject of cataract tenderly touches a foible of our human nature, which expediently, for all concerned, it is best to avoid exciting. Again, the following incident shows the effect which a similar premature announcement produced upon a member of our own sex: In the smoking-room of a large London club the question of cataract was being discussed. The matter arose in consequence of a well-known member having recently been operated upon for the disease. In the course of the general conversation, one of those present dilated upon his own experience; he had been told, he said, by an ophthalmic surgeon, mentioning the name, that he had cataract, "and I don't believe him," he added, "because that was two years ago, and I see just as well now as I did then, whereas, had he been right, I ought now to be blind." But our common human nature may find still another expression in regard to cataract, of which the following example is worth mentioning: a middle-aged, wealthy spinster, now no longer living, who had, during her later life, succumbed to the elusive charms of rejuvenation by art, became the subject of cataract. When the time arrived for operative interference she paid visits to various ophthalmic surgeons before making up her mind upon the question of the operator. At length at one visit she was informed by her adviser that she had "the most beautiful cataract he had ever seen." She was aware that she had passed the age of beauty; but her cataract was beautiful, and its beauty was natural. It was worth something to be told that she was the possessor even of a cataract which was beautiful. Naturally the appointment for the operation was arranged forthwith.

The second class of cases in which so-called second sight may occur is of much more practical interest to the ophthalmic surgeon than that which we have just considered. It comprises those cases of spontaneous cure of cataract, of which a goodly number has now been recorded.

The methods by which the spontaneous cure of cataract can be brought about are two in number, each of which is dependent upon the secondary processes of degeneration associated with the pathological changes occurring in the opaque lens. The first is that in which the lens undergoes spontaneous dislocation, the second that in which intra-capsular absorption ensues, after liquefaction, of the opaque lens fibres. Of these two classes of cases, the latter class occurs with greater frequency, the literature of the subject now containing a goodly number of recorded cases illustrative of the changes in question.

Let me first, however, speak of the former class, of which I shall narrate two or three interesting examples which have come under my own observation.

The lens, as you are aware, is held in position in the eye by its suspensory ligament, otherwise

called the zonule of Zinn. The zonule is really the prolongation forwards of the hyaloid membrane. Opposite the ora serrata this membrane assumes a firmer consistency, approaching in character that of elastic and connective tissue; it afterwards divides into two layers, the anterior of which is prolonged forwards, and is firmly inserted into the anterior capsule of the lens, while the posterior forms the support for the lens behind. It follows, therefore, that in any case in which the lens becomes dislocated, some change has ensued by which the lens, wholly or in part, has lost the support of its suspensory ligament. So far, however, we do not possess any definite information as to the exact changes which lead to the spontaneous displacement of a cataractous lens. On the other hand Becker has ingeniously suggested that the displacement may directly follow the evolution of secondary changes in the lens capsule, which ultimately lead to rupture of the zonule. In the formation of capsular cataract, in association with an over-ripe cataractous lens, a cellular infiltration occurs upon the inner side of the capsule. This infiltration, proceeding further in its organisation, in the course of time commences to contract, and eventually results in dragging away the capsule from the fibres of the zonule, thus releasing the lens from its zonular support. When this process has become complete, spontaneous dislocation downwards ensues, and the lens lies free in the fossa patellaris of the vitreous. This means, of course, to the cataractous patient in whom the eye is otherwise healthy, the sudden and complete recovery of vision. In a sense the event is a miraculous one to the patient. The saying of the blind man of old could be equally applied in these cases: "One thing I know, that whereas I was blind, now I see."

Not by any means the least interesting feature of these cases is the circumstance under which the spontaneous dislocation occurs. Norris, in his exhaustive article upon this subject in Norris and Oliver's 'System,' refers to several cases in which the special circumstances are described. Allusion may be made to some of these. In Desgrange's case, for example, recorded in 1831, a peasant, in whom one lens had been cataractous for nine years, was one day thrashing in a barn. Suddenly he felt a curious sensation in the eye, and immediately afterwards he found that the sight had been recovered, owing, as it was afterwards shown, to spontaneous dislocation of the lens. Again, in a case described by Fischer, the patient happened to be applying to his cataractous eye a solution of "lapis divinus with Sydenham's laudanum," whatever that may be, and just at the moment of application the lens became displaced downwards, and the vision was restored. It is recorded that the patient ever afterwards "kept the precious receipt, as if it were gold, and recommended it to all blind people." Another curious case was one recorded by Recordon. The patient, an old man, was admitted into the hospital to undergo the extraction of a cataract. The operation, however, had to be postponed in consequence of an attack of rheumatism, to which disease he was subject. After some days he noticed that while lying in bed he could see the upper part of the window-frame opposite, and that this vision was lost when he sat up. In about eight days it is recorded that the vision was perfectly recovered, and it was found that the lens had disappeared wholly into the vitreous.

The following are the notes of an interesting case which came under my own observation. On October 15th, 1901, a healthy-looking, single lady, æt. 59 years, was sent to me in consequence of the failure of her vision. She gave the history that fifteen years previously she had consulted an ophthalmic surgeon, who informed her that "she had a tendency to cataract." I found well-marked stellate striation of each lens—more apparent in the left eye. No lens improved the vision for distance—$\frac{6}{0}$, but with + 5 sph. she could read J. 2 at 6 in. Once a year she subsequently saw me, the lental opacities gradually developing until the vision failed for reading. But as she still retained sufficient vision for seeing her way about she expressed the desire to postpone operative interference. In October, 1908, she paid me another visit. At this time she stated that, as far as she could judge, the sight was no worse than it was a year previously, although it was now two years since she had been able to read. With the right eye she could count fingers easily at 3 ft., and with the left at 9 in. At this visit she agreed to extraction of the left lens, provided that a little time was permitted her to make some necessary arrangements. It was consequently arranged that she should return in a month's time, and meanwhile I ordered a 1 gr. solution of atropine to be applied

twice daily to each eye. At the appointed time she returned and made the following statement: "Twenty-four hours after using the drops I suddenly found that I could see quite plainly with each eye." Examination of the eyes showed that each lens had dropped below the centre of the pupillary area, leaving the upper two thirds of the pupil clear and black. The position of the lenses was indicated by their upper opaque elliptical margins—that is to say, spontaneous dislocation downwards had occurred. The distant vision in each eye corrected with + 10 was $\frac{6}{9}$, and $\bar{c}$ + 14 the near vision was J. 1 at 8 in. Neither eye showed any reaction; the tension was normal, and there was no history of traumatism. In order to determine how much of the recovered vision was due to dilatation of the pupil, the atropine drops were discontinued, and subsequently it was found that the difference between the vision, with the drops and without, was practically *nil*. So far the patient has remained in the perfect enjoyment of her recovered vision; no untoward symptoms have developed, and time alone can determine whether changes of a destructive nature will supervene in consequence of the abnormal position of the lenses. It can be well imagined what the consequences would have been had such a case as this fallen into the hands of a quack, with the result as described. Blazoned forth in the advertisement columns of the press, in large and bold type, would have appeared a full account of the marvellous recovery of the sight, without operation, in a patient blind from cataract. On the strength of his statements, which, of course, would be unassailable, his premises would be quickly besieged by rich and poor alike, all seeking the aid of his marvellous skill. Throughout the length and breadth of the land his name would be pinnacled as the great curer of cataract without operation. Ophthalmic surgeons would be woefully placed at a discount; no longer would the public believe that an operation for cataract was necessary. "Use my drops and only have patience," would be the quack's advice, "and your sight will be recovered without any operation." And such a contingency is by no means impossible. Nature, we may suppose, holds no special brief for ophthalmic surgeons. But, humanly speaking, it would not be "cricket," nor would it be fair on the part of Nature to place such power, for deluding the cataractous community, in the hands of an unauthorised, uncredited, presumptous disciple of Hippocrates. Even so it happened with my patient. Her friends having found how her sight had been recovered, many applications reached her for a copy of the prescription of the drops. Gifted, however, with good common sense, and mindful of the inexpediency of granting such requests, she resolutely declined to act the part of a cataract curer without operation.

But spontaneous dislocation of the cataractous lens may assume a different form to that just described. A man came to my out-patient room some years ago complaining of failing vision in the left eye. This was found to be due to incipient cataract. He did not return for five years; the left eye was then slightly injected and painful, and in the anterior chamber was lying the completely opaque lens. He was at once admitted into the hospital for extraction, but at the time of the operation it was found that the lens had slipped back into the vitreous. Directions were therefore given to get the patient to move his head about until the lens reappeared in the anterior chamber, and then to cut off its retreat by contracting the pupil with eserine. In a few days this necessary result was accomplished, and I effected the extraction of the lens easily, with the recovery of vision.

There is, however, yet another side to the picture of these cases of spontaneous displacement of the lens in cataract. The results are by no means all satisfactory. Indeed, it is well known that a lens, wholly or in part detached, whether cataractous or not, is a source of danger to an eye by exciting various intra-ocular changes, such as a foreign body is liable to produce. When located in the vitreous, a lens tends to break down and disorganise that structure owing to its continual oscillation with the movements of the eye. It tends also to disturb the nutrition of the ciliary processes and choroid, by which these structures may pass into an atrophic condition. Furthermore, there is always some risk of a displaced lens becoming the *fons et origo* of an acute glaucomatous attack. In some cases a partial dislocation of a hypermature cataractous lens may occur, which, by compressing the angle of the anterior chamber, causes a chronic glaucomatous process to supervene, as the result of which the vision is ultimately totally and permanently destroyed. This is one of the contingencies to

which patients with hypermature cataracts are subject. The insidious onset of the glaucoma is overlooked and its progress ignored, the gradually increasing loss of all vision being attributed to the cataract. Then at length the ophthalmic surgeon is seen with a view to the extraction of the opaque lens, and the discovery is made that the eye is totally blind. The following case, however, had a more favourable termination: An old woman, æt. 79 years, was sent to me at the hospital suffering from an acute attack of glaucoma in the left eye of three or four days' duration. For twelve years the eye had been blind from cataract. I made a large upward section, together with iridectomy, and extracted a hyper-mature lens; this relieved her glaucoma, and she recovered vision to $\frac{6}{12}$, and J. 1 with correction.

I have now to speak of the second class of cases of spontaneous cure of cataract—those in which intra-capsular absorption occurs of the opaque lens matter. According to Fuchs, "this takes place (*a*) by the absorption in exceptional cases, not only of the cortex, but also of the nucleus, to such an extent that nothing but slight opacities remain; (*b*) by the formation of a Morgagnian cataract and the subsequent transformation of the blind portion of the cataract into a clear, transparent liquid." In these cases the upper part of the pupil is black, while the brown nucleus is seen lying in its lower part. Afterwards, the transparent liquid and even the nucleus itself may be absorbed, so that only a thin capsular membrane is left. "(*c*) By spontaneous dislocation of the lens, so that the pupil again becomes partly or entirely black."

Of the large number of cases now recorded in which these intra-capsular changes have taken place, the case by Solomon—the ophthalmic surgeon, not the king of that name—in 1858, was the first to be published. It was a case of an infant at the breast, in whom spontaneous intra-capsular absorption occurred of congenital cataracts, in the course of a month's treatment with atropine. So far as I have been able to ascertain only two other cases of the kind have been recorded—those by Kolscher. In these the absorption processes occupied a much longer period, the pupils not becoming clear until the end of five years.

Various results in the improvement of vision have been obtained in adults following these intra-capsular changes; in some almost full vision has been attained, while, curious as it may seem, in others Nature has done better than the surgeon, for several cases have been recorded where a patient has submitted to cataract extraction in one eye, while later on spontaneous absorption of the opaque lens has occurred in the other, with the recovery of better vision than that which the surgeon had obtained in the eye submitted to operation.

Analysing these cases of spontaneous dislocation of cataractous lenses, and the spontaneous intra-capsular absorption of cataracts, we find certain differences between the two, to which attention may now be directed. In the first class the recovery of vision is practically instantaneous and perfect, but the change is confined to cataracts of long duration; and moreover, owing to the abnormal position of the lens, risks are induced of glaucoma and of other disorganising effects upon the eye. While, therefore, the immediate result may be miraculous, and excellent, of spontaneous dislocation of the lens, there remains the possibility that at any time, under such circumstances, destructive changes may be excited. On the other hand, there appears to be no limit of time in which a cataractous lens may commence to undergo spontaneous intra-capsular absorption. For example, Lange records a case in which a cataract took four years to mature, and three years later began to be absorbed. Naturally in these cases the recovery of vision is a gradual process, depending both upon the rapidity with which absorption takes place and the degree of transparency of the capsule. Indeed, some years may elapse before complete absorption is accomplished. But in these cases the integrity of the eye is for the most part not imperilled. Although glaucoma and detachment of the retina have been met with, any complication of the kind, as recorded cases have shown, is quite exceptional, and probably when occurring, is dependent more upon a pre-existing tendency rather than upon one directly due to the secondary changes of which the cataractous lens is the seat.

The facts which I have thus endeavoured to lay before you deal with, I think, an interesting aspect of ocular disease. So-called "second sight," although a well-recognised condition, is nevertheless not one about which the ordinary text-book has much, if anything, to say. Presumably the reason of this may be that the cases do not occur with sufficient frequency to render it needful to devote space to their discussion. And yet, as we have seen, the subject contains many details worthy of special consideration, details which are both interesting and instructive, the knowledge of which is calculated at any time to prove useful and profitable, not only as a means for educating in this regard the popular world as to the difference between miracles and science, but also as a means by which the practitioner can safeguard himself against the pronouncement of an opinion that every case of cataract demands for its relief operative interference.

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CONTENTS.

PAGE

***Two Clinical Lectures on Hemiplegia and its Treatment.** By GORDON HOLMES, M.D.Dubl. LECTURE I ... 129

***Three Clinical Lectures on the Heart.** By SAMUEL WEST, M.D., F.R.C.P. LECTURE III 137

***The Hyperæmic Treatment of Inflammation.** By A. V. POYSER, M.B., B.C. 139

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TWO CLINICAL LECTURES

ON

HEMIPLEGIA AND ITS TREATMENT.

Delivered at the National Hospital for the Paralysed and Epileptic.

By GORDON HOLMES, M.D.Dubl.,
Assistant Physician to the Hospital.

LECTURE I.

GENTLEMEN,—It may possibly appear to you to be unnecessary to devote two lectures to hemiplegia as I propose to do, but on the one hand hemiplegia will be one of the forms of nervous disease with which you will meet most frequently in practice; and in the second place there are many points in the nature and treatment of hemiplegia which I think it is profitable to consider.

By hemiplegia we mean loss of power in the limbs of one side, and to a less extent in the muscles of that side of the body, resulting from a lesion of the tracts which connect the brain, or rather the cerebral cortex, with the spinal cord, in other words of the upper motor neurons. Here I have drawn a rough diagram of the pyramidal tract; if you follow on it the course of the fibres which constitute the pyramidal tracts, you will find that they take origin in the cerebral cortex, and pass down through the posterior limb of the internal capsule and through the mid-brain, pons and medulla; in the lower part of the latter they decussate almost entirely to pass into the opposite side of the spinal cord. Therefore the pyramidal tracts which take origin in one hemisphere of the brain terminate in the opposite side of the spinal cord, and there transfer the impulses which they carry down to the lower neurons, or the motor nerves that take origin in the anterior horns. Therefore, interruption of one of the pyramidal tracts above its decussation will produce

paralysis of the opposite side. Hemiplegia may result from destruction or interruption of these fibres in any part of their course. The lesion may be in the cortex, in the sub-cortical white matter, in the internal capsule, in any part of the brain-stem, or even in the upper portion of the spinal cord, and in any of these regions will produce the typical picture of hemiplegia. We have now fairly definite evidence of the exact site that a lesion must occupy in the brain to affect or destroy the pyramidal tracts, for we know that these tracts take origin only from the cortex immediately in front of the fissure of Rolando, from the greater part of the ascending frontal or pre-central gyrus, but towards the apex of the hemisphere from a part of the superior frontal convolution as well, and on the mesial surface from part of the paracentral lobe.

Hemiplegia as a clinical entity may be considered from various points of view. In the first place, I might devote a considerable portion of this lecture to dealing with its causes, but this I do not propose to do, as I think other aspects are more interesting and more instructive. In the greater number of cases it is due to a vascular lesion, either a hæmorrhage from a ruptured vessel, or a softening due to thrombosis or embolism of one of the vessels that supplies an area through which the pyramidal tracts pass, or it may be due to a tumour compressing or destroying them. From the clinical point of view it is important to determine what is the cause of hemiplegia, but from the point of view of treatment this is less important than other aspects of the condition.

I intend in these lectures to deal with the symptoms of hemiplegia. They vary from case to case. In the first place you may have the symptoms varying with the position of the lesion. For instance, a lesion in the right side of the pons may interrupt the pyramidal tract which passes to the left side of the spinal cord, and consequently produce paralysis of the left limbs, and at the same time it may involve the seventh nerve that supplies the facial muscles of the right side and so produce an alternating hemiplegia, that is, paralysis of one side of the face of the peripheral type, and of the limbs on the opposite side. Or a lesion in the right side of the mid-brain may produce paralysis of the left limbs, and at the same time destroy part or the whole of the oculo-motor nerve of the right side. Then you would have right ophthalmoplegia associated with left-sided hemiplegia. While a lesion in the medulla may produce paralysis of one glosso-pharyngeal, or vagus, or hypoglossal nerve, and of the limbs on the opposite side. On the other hand, the symptoms may vary according as centres other than the pyramidal tracts are also involved, which may modify the picture of the condition. In the internal capsule the motor fibres of the pyramidal tracts pass between the optic thalamus and the corpus striatum. If the optic thalamus, for instance, is involved, we have a remarkable picture, which I hope to show you in the next lecture; and the condition of tone of the paretic muscles is quite different when the lesion lies in the pons to that which obtains when it lies in the higher levels of the brain. In considering the symptoms which result directly from interruption of the pyramidal tracts, we need only distinguish between lesions of the cortex and lesions of the subcortical portion of the pyramidal fibres, for we believe that once an impulse is started in the cortex it is probably carried unchanged to its destination in the spinal cord. But certain differences in the clinical picture may result according as the lesion is in the motor cortex or in some part of its efferent tract, as this diagram will make evident to you. In and near the cortex the fibres for the different regions are widely separate anatomically, for instance the fibres which go to innervate the leg come from the upper portion of the hemisphere, those for the arm from the middle portion, and for the face from the lower portion, so if the lesion be small it may affect only the arm or the leg centre and cause monoplegia. But a lesion of the same size in the internal capsule may interrupt all the fibres of the pyramidal tract, and so produce a complete hemiplegia. A greater tendency to produce limited paralysis is characteristic of cortical lesions, while in the internal capsule and below it the motor fibres are gathered together in a compact strand, so that a lesion in it produces hemiplegia, with relatively little regional variation in the paralysis of the different limbs.

I show you now a man, æt. 25 years. When nineteen, that is to say, six years ago, he had a sudden stroke; its cause is easy to ascertain, as he has severe morbus cordis with a double mitral murmur, and he has since had other signs of cardiac affection. For a time after the stroke his left

limbs were almost completely paralysed, but since then he has gradually improved. If we examine him systematically now we find all his ocular movements intact, and that his pupils react well to light and on accommodation. His jaw descends straight as he opens his mouth, and if you feel the masseters as he clinches his jaws you find that the muscles on the two sides contract equally. As he attempts to show his teeth he separates his lips a little wider on the right side than on the left, but there is no difference in the contraction of the frontalis on the two sides, and the muscles for frowning contract equally on both sides. The tongue is now protruded straight, but for a time it deviated to the paralysed side, and his palate moves symmetrically. His power of movement is still seriously reduced in the left arm, and most movements are restricted in range. He is unable to place his left hand on the top of his head; he can bend the elbow only slowly and with great effort; he cannot open his fingers fully, but he can flex them up to some extent. The movements about the shoulder joint are much less affected; he can perform many of them fairly well. We find much the same condition in the left leg. He does not succeed in straightening the knee completely, and he cannot flex it beyond the right angle. Movement of the foot is even more limited; the power of dorsi-flexion is almost totally lost, but he is able to extend his foot; his power of moving the toes is practically absent. You see at once that the degree of affection of the different muscles of the hemiplegic limbs is very variable. Further, as he breathes there is practically no difference in the movement of the two sides of the chest, and in bending or straightening his back he contracts the spinal and abdominal muscles equally on the two sides, so the trunk muscles are little if at all affected. We have found that the orbicularis oculi, the frontalis, and the corrugator, too, escape on the left side, and he has also no difficulty in swallowing, and his power of phonation and articulation, that is to say the functions of the pharyngeal and laryngeal mechanisms, are practically unaffected. Finally, the masseters and the ocular muscles contract equally on the two sides. What is the explanation of this relative escape of some muscles? The explanation was expressed by the late Sir William Broadbent, in the law which bears his name, that muscles which are bilaterally innervated and which are generally employed simultaneously on both sides of the body escape, or are very little affected in hemiplegia. We wrinkle our foreheads on both sides at once, and we move both sides of our chest together, and if we attempt to contract one masseter we cannot help contracting the other as well. The second disparity in the palsy in the muscles is that in both the arm and leg he is able to execute movements about the proximal joints much better than about the distal. There is practically no movement of the left fingers, toes or ankle, whereas there is a fair range of movement of the shoulder and an excellent range of movement at the hip But in every case we find there are other muscles or groups of muscles which are very severely affected. Most hemiplegics find it very difficult to rotate the shoulder outwards, and the extensors of the fingers suffer much more than the flexors; and in the leg you generally find the flexors of the knee and the dorsi-flexors of the foot much more paralysed than the extensors of the knee and foot. In cases where a certain amount of recovery has occurred, we see another very common feature of hemiplegia, which is that though the patient may be fairly well able to execute large and coarse movements, he cannot perform finer or isolated movements—for instance, this second patient I show you can easily flex all his fingers together, but he is unable to flex any one alone. The explanation of this is probably that it is not the cortex alone which is concerned in volitional movements. You will remember that in the lower vertebrates no cerebral cortex exists, or is at least certainly not used in the initiation of volitional movements; and you must be aware of the fact that in certain animals—the dog, for instance—the whole cerebral cortex can be removed and yet the animal may move about and perform movements almost as well as a normal dog. It seems very probable that the subcortical centres that can initiate movements under these conditions may not be entirely latent in man, and that through them some of the larger and coarser movements that hemiplegics can perform may be produced. But the movements which can be performed under such conditions are in the first place only gross general movements; the patient may be able to shrug his shoulder, throw the arm about, or swing the leg about in a rough way, but the power of

executing finer movements is completely gone. In such cases, too, associated movements are easily produced; you see that when he grasps my hand or any other object firmly with his normal hand, or attempts to perform any other movement of this limb against resistance, the paralysed limb generally moves involuntarily; these involuntary movements are sometimes similar to the purposive movements of the sound limb, but in some cases only flexion of the fingers or of the elbow can be observed.

The character of gait is another important feature of hemiplegia. The paretic limb is kept fully extended or almost so as he walks, and is swung forward from the hip as a rigid piece. You notice, too, that it does not move in the sagittal plane of the body in the ordinary way, but is circumducted. If you examine, as you must in all these cases if you wish to treat them properly, the causes of this affection of walking, you will find the chief trouble is that the patient cannot raise the leg, so that the foot must be dragged along the ground on which he walks. That is due partly to the phenomenon which we shall have to deal with next, contracture or rigidity, which prevents full dorsiflexion of the foot and adequate flexion of the knee. Another striking phenomenon is the peculiarity of lateral walking. This man was aware of it himself, and has tried to correct it. He has right-sided hemiplegia; in which direction will he find it easier to progress sideways? As a matter of fact we generally find that it is easier for the hemiplegic to walk lateralwards with the affected side in front; the reason is that, as he attempts to move either foot forward, his centre of gravity follows it, and he must consequently tilt his pelvis towards the opposite side to keep the centre of gravity on the foot on which he stands, and he can naturally raise the affected foot from the floor more easily when the pelvis is tilted towards the opposite side. You will readily understand that the difficulty in raising the palsied foot from the ground is greater when he attempts to walk with the sound limb forwards. On the other hand, in functional or hysterical hemiplegia the patients invariably find it much more difficult to walk with the affected limb forwards. Another sign in organic hemiplegia was originally pointed out by Babinski; if your hemiplegic patient is lying down and you ask him to rise from the supine position without the aid of his hands, you find that the palsied limb rises from the couch in the effort, while in hysterical hemiplegia the affected limb does not rise at all.

We find that in most cases of hemiplegia there is no definite reduction in the size of the muscles, but where the hemiplegia has lasted for long the muscles of the affected limbs may be somewhat smaller than those of the other limb. But the wasting, as a rule, is not more than can be explained by the inactivity of the limbs; you can understand that if the muscles of a limb are not used for a long time they must diminish in size. Occasionally, however, we find a considerable reduction in the bulk of the muscles, so great sometimes that we can speak of a true atrophy; it is then generally the small hand muscles which are most affected. Many theories have been put forward to explain this muscular atrophy in hemiplegia, for, as the text-books tell you, one of the characters of an upper motor neuron palsy is that the muscles do not waste or exhibit any change in their electrical reactions. One view attributes the wasting that occasionally occurs to a vaso-motor paresis, and it is true that in most cases of hemiplegia the affected hand is much colder than its fellow, and is often puffy or even œdematous compared with the normal hand. Others have ascribed the slight wasting of the muscles which occurs to the arthritic changes that developed frequently in hemiplegia. I do not think that is a possible explanation, as in my experience the muscles which waste are muscles which are not related to the joints which are usually most affected; in the arm for instance, it is the shoulder-joint that is most frequently and severely involved, whilst it is the smaller muscles of the hand which waste as a rule. It seems probable that even the upper motor neurons exert a certain trophic influence on the muscles to which they are functionally related, and we have the anatomical fact that occasionally in hemiplegia, in cases which come to necropsy shortly after the onset of the lesion, definite changes may be found in the anterior horn-cells that innervate the palsied muscles.

Now I pass to the subject to which I intended to devote the greater part of this lecture; it is the feature of hemiplegia which is most important from the point of view of treatment—this is the phenomenon of increase of tone, or rigidity, in the affected muscles. Take this young man's arm and you will find that it is difficult to straighten these

fingers by passive movement, but if you persist long enough you can succeed in doing so. There is obviously an increased resistance to movement and the stretching of the muscles that it necessitates. You find exactly the same phenomenon at the elbow and the other joints of the limbs; even movements of small range are difficult. This is what we know as rigidity or hypertonia of the muscles; it consists in a tendency of the muscles to approximate their points of origin and insertion to one another. In hemiplegia we meet with two different forms of rigidity. The first is the early rigidity that follows immediately the onset of the attack and passes off generally in a few hours, certainly within forty-eight hours. This early rigidity is generally found in diffuse cortical lesions, for instance, hæmorrhage over the surface of the brain, or whenever hæmorrhage bursts into the ventricles. The other variety of rigidity is the late and persistent contracture that follows on degeneration of the pyramidal tracts. You find that the range of all movements is limited and the rigidity is permanent. It is not, however, always constant in degree; in such a case as this you would find that when the patient was asleep it is very much less, and in that condition, or immediately on waking, it would be easier to extend his fingers or perform passive movements. In the early morning, too, it is always much less than later in the day; the patient, in fact, volunteers the statement that in the early morning he can easily open his fingers, which you see he is now unable to do. Further, if you placed his hand in a warm bath, you would find the rigidity gradually diminishes, and that you could open the fingers with very much less power; and if you wind an Esmarch's bandage round the limb tightly the rigidity would almost completely disappear. Therefore this rigidity or hypertonicity of the muscles is not constant, but varies under different conditions. This is one of the most prominent features of hemiplegia, and I think it is the one to which we should devote more attention than we do in dealing with the treatment of such cases. We therefore have to consider, as shortly as we can in the time allowed, what is the nature and cause of this rigidity. Various theories have been put forward, but all assume that the destruction of the pyramidal tract removes an inhibiting action of the cortex upon some subcortical centres which have a tonic effect on the muscles, and consequently when uncontrolled by the cerebral cortex produce an increase of tone or rigidity in the muscles which are paralysed. Consequently there is rigidity where you get a degeneration of the pyramidal tracts, and the letting loose or unbridling of the subcortical centres. But matters are not quite so simple as that, because in the first place if you examine this boy, or any other case of hemiplegia, you will find that this contracture tends to hold the limb in certain positions, as the rigidity is greater in some muscles than in others. Why? Mann's theory was for a long time accepted. He says that on the one hand, where a group of muscles and their antagonists are paralysed, it is always in that group which is least weakened that the contracture is most marked, as the greater paralysis of one muscle permits shortening of its antagonists. He has insisted that where there is complete paralysis of a limb you cannot get increased rigidity, and where there are groups of muscles equally paralysed you must not expect to find contracture fixing the limbs in any one position, as the antagonistic groups remain balanced. But we may get rigidity in limbs in which there is no power of voluntary movement; we rarely meet with it in hemiplegias, but we see it frequently in spastic paraplegia, in which there may be enormous rigidity and yet no power of voluntary movement. Förster, whose work has not attracted as much attention in England as it deserves, adopts another explanation. He points out that it is the tendency of every muscle, whether normal or pathological, to tend to approximate its points of insertion, or to shorten itself as much as possible; this is what we mean by tone, and when the points of insertion and origin of muscles are approximated they tend to adapt themselves in length to this shortening by a gradually increasing development of this tone. And this tendency becomes greater when the inhibiting influence of the cerebral cortex is removed, and thus it produces rigidity. If this be so it is a necessary corollary to his views that the position in which the palsied limb is allowed to lie must have a very great influence upon the form of contracture which develops in it. Therefore if you keep the palsied limb straight there will be a condition rarely seen in hemiplegia—an extensor contracture of the limb; Förster has shown that it may occur. But when a man gets hemiplegia he is put to bed and the arm is placed across the chest with the elbow flexed. And even when he

leaves bed he finds it troublesome to have the arm, which is a dead weight, hanging by his side, and consequently supports it in a sling, or fixes his fingers into his clothes, with the elbow flexed. This, I believe with Förster, is the explanation that in hemiplegia there is usually flexor contracture of the upper limb. On the other hand, a patient in the hemiplegic state will naturally lie in the bed with the lower limb extended, and so the contracture of the lower limb is generally of the extensor type. But if the leg is kept flexed the developing rigidity may fix it in that position. Therefore, as we find that the position in which the limb is allowed to rest has a very great influence on the development of contracture, it is evident that if the limb is not allowed to remain constantly in any one attitude, but is frequently moved about, there is less danger of contracture fixing it in one position. And that is so. There is, perhaps, no better illustration of the truth of Förster's views than that furnished years ago by Munk's observations. He found that after removal of the motor cortex in monkeys contractures did not, as a rule, develop as they do in man. But his monkeys were kept in a large room and permitted to move about freely. If, however, any of these animals after extirpation of the motor area was kept confined in a small cage and they were allowed to sit stationary, in a short time contractures developed just as in man. One reason that contractures do not develop so strongly or readily in the lower animals after lesions of the pyramidal tracts as in man is that even after complete destruction of these tracts they preserve much more power of movement than man, and the palsied limbs consequently do not remain fixed in any one position.

But though position may be the most important feature in determining contracture, there is undoubtedly a greater tendency in some muscles to contracture than there is in others, and no matter what you do you will find the flexors of the arm and fingers become more rigid than the extensors. We have already seen that the rigidity of the limb is not constant, but varies under different conditions, and if you examine your cases carefully you will find that it is increased by any form of irritation or stimulation of the limb. These patients I show you will tell you that as soon as the affected limbs become cold they become more rigid and they cannot move them as well. And if you stimulate the skin in any way you will see that the rigidity will gradually increase. And this explains the pernicious influence that a faradic battery has on the course of many of these cases when the treatment is not carried out under proper supervision. We find other evidence of this fact in both experimental physiology and in clinical work, for I may remind you that Professor Sherrington has shown that after section of the posterior roots of the spinal cord, and consequently the exclusion of afferent impulses, rigidity, however it was produced, disappears in the corresponding limb. And we find that where tabes and hemiplegia are associated, if there be much degeneration of the posterior roots, rigidity does not develop, or it is much less than it would have been under ordinary conditions. Therefore by eliminating as far as possible unnecessary afferent impulses we may expect a considerable reduction of the contractures or rigidity.

Rigidity may have a very serious influence on the voluntary movements that are possible; this young man, for instance, when the contracture is less, can open his fingers and move them fairly freely, but when the rigidity increases he cannot move them at all. Thus rigidity limits the power of voluntary movement of the limbs.

In the second place, when there is a less degree of paralysis rigidity disturbs the co-ordination of the various muscles concerned in the execution of any action. Here is a man in whom there has been a considerable return of power in the arm, but you see that as he attempts to touch my finger with the affected hand his movements are slow and awkward, evidently in large part due to difficulty in relaxing the antagonists of the muscles he must contract in performing the movement. He is a school-master, and though he can now write fairly well with a pencil, he is practically unable, owing to this difficulty, to draw or write on the blackboard with chalk.

We have dealt so far only with the condition known as spastic contracture, which is merely an increase of tone in the muscle which tends to shorten it, and offers a resistance to its passive extension. But in many of these cases you will find that it is impossible to stretch the muscles to their normal length even under an anæsthetic This cannot be due only to increase in tone of the muscles, for if it were we should be able to over-

come it. It is a condition which follows frequently severe spastic contracture, and is known as organic contracture, or *rétraction fibreuse*; it results from structural changes and the deposition of fibrous tissue in the palsied muscles, which consequently lose their elasticity. Whilst with spastic contractures we find the deep reflexes much exaggerated, in organic contractures those which are subserved by the palsied muscles may be absent. The development of organic contractures produces a condition which Dr. Thomas Buzzard pointed out as an important diagnostic sign between organic and hysterical hemiplegia. You see I can succeed in bringing this boy's fingers into the same plane as his hand, and I can extend the wrist till the hand lies in line with the forearm, but I cannot bring the forearm, hand and fingers at once into the same place, as if the wrist be extended a greater elongation of the flexors of the fingers is necessary to straighten them than if it be flexed.

Certain involuntary movements occasionally occur with hemiplegia in the form of tremor, athetosis, or choreiform movements, but we have strong evidence that none of them result from a pure lesion of the pyramidal tract; a few cases have been reported in which a lesion of the cortex was said to have produced choreiform movements, but this is doubtful. We shall deal with these spontaneous movements in our next lecture. A form of clonus, which may be excited by suddenly putting the hypertonic muscles on the stretch, may occur in a severely paralysed limb, and it is the only variety of spontaneous movement which occurs in pure hemiplegia.

One of the most important signs of hemiplegia is a change in the reflexes on the paralysed side. We find the pupillary reflexes invariably intact, except when the lesion lies in the mid-brain and injures the third nerve nucleus. The deep reflexes, as the knee-jerks, are a measure of the tone of the muscles, and can be elicited from the tendons of various muscles. These reflexes are exaggerated on the palsied side; thus, tapping on the triceps tendon evokes a much stronger extension of the elbow than in the sound side, and the reflex flexion of the elbow, which may be elicited by striking the lower end of the radius, or by a sharp blow on a finger placed directly in the biceps tendon, is also greater on this side. The knee-jerk, too, you see, is much increased on the paralysed side. We take the ankle-jerk by striking the Achilles tendon directly; on the affected side there is not only a brisker response, but ankle clonus is easily produced; you see I can elicit this in the ordinary way, too, by suddenly dorsiflexing the foot. The most important of the superficial reflexes with which we have to deal in such cases are the abdominal and epigastric reflexes; these are elicited by stroking the skin over the lower margin of the costal arch, or over Poupart's ligament. On the normal side the reflex contraction of its muscles draws the abdominal wall to the side stimulated, while on the other side stimulation, as you see, produces no effect. Another and more important superficial reflex is obtained by stroking the sole of the foot: on the normal side you see stimulation of the sole by a blunt instrument leads to flexion of the great toe, but on the paralysed side the great toe extends and there is some spreading of the other toes. This is the extensor type of the plantar reflex, or Babinski's sign, which, when it occurs, is unequivocal evidence of a lesion on the pyramidal tract.

The reflex functions and the voluntary control of the sphincters are as a rule not disturbed in hemiplegia.

Finally, we must remember there are other conditions apart from paralysis of the motor system and the reflex changes associated with it which may result from such lesions as produce hemiplegia. Sensory troubles, too, may occur, but when only the cortex or the white matter immediately below it is destroyed, the ordinary forms of cutaneous sensibility are little affected. The patient can usually feel the slightest touch, as you see in this boy, on the one side as well as on the other, but there is frequently a defective knowledge of the position which the paralysed limb occupies in space, and a difficulty in recognising objects placed in the hand by their shape or form, and in discriminating between one point and two points of Weber's compasses. Vision is the only one of the special senses which is much affected in hemiplegia; when the lesion lies in the internal capsule and extends to its posterior part it may interrupt the optic radiations and produce hemianopia of the hemiplegic side.

The scope of this lecture will not permit us to consider the various forms of disturbance of speech that may be associated with hemiplegia, but it is

necessary to refer shortly to *apraxia*, which is also occasionally found in cases of right-sided hemiplegia, since if it is overlooked its symptoms may give rise to considerable confusion. Apraxia is a condition in which the patient, though able to perform certain elementary or simple movements, cannot combine them and direct them to a definite purpose or for a particular action. Though there may be good power of movement in the affected limb, you may find the patient unable to beckon with it, or to shake hands, or to knock on a door, or turn a key. You can readily demonstrate it by handing your patient a matchbox and a candle, asking him to light the latter; the apraxic patient will be unable to do so, though he understands your request fully; he may fail in any stage of the procedure, either in opening the matchbox, taking out a match, in striking it on the box, or even in bringing the lighted match to the candle. The subject of apraxia has become so complicated and has undergone so many subdivisions that a considerable time would be required to deal with it adequately. It seems to be due, as Liepmann has pointed out, to interruption of the connections between the cortical centres for the execution of voluntary movement and the sensory centres that lie around them. It must be evident to you that if you fail to recognise its nature you may have great difficulty in understanding and interpreting the symptoms which the patient exhibits.

As we have now seen the chief features of hemiplegia and have considered their nature we can satisfactorily deal with its treatment. The first aim in the treatment of every form of disease is to remove its cause, but in hemiplegia in the majority of cases, except in those in which it results from a tumour or other localised lesion that compresses or indirectly affects the motor cortical centres or the pyramidal tracts, this can be rarely attained. It is not my intention to refer to the therapeutics of the early stages of hemiplegia, in which the chief aim should be to treat the vascular disease to which the lesion is generally due, but to limit my remarks to the treatment when it is developed. We have no evidence that we can in any way modify or influence the actual lesion, for brain tissue, if once destroyed, does not regenerate; recovery can consequently occur only if the destruction is incomplete, or if undamaged portions of the brain or other tracts take on some of the functions of those that have been destroyed. Consequently our treatment at this stage can be only symptomatic.

That considerable recovery from the condition that exists immediately after the stroke may occur is well known to you, and the two cases I have shown you illustrate it very well. We must consider in what way we can aid or supplement this tendency to recovery. The chief symptom is paralysis of voluntary movement, and unhappily we can do nothing to influence this directly, but, as I have shown you, when power begins to return the use of the limb may be seriously restricted by rigidity and contractures, as well as by the joint changes that tend to develop. And the rigidity not only limits the range of movement at each joint, but the defective inhibition of the tone of the antagonists of the contracting muscles makes the movements that the patient becomes able to execute slow, awkward, and inco-ordinate. If, then, we can diminish the tendency to rigidity and prevent the development of contractures and adhesions in the joints, we can do much for our patients. And we can do much in those cases in which the lesion is not so extensive as to make some recovery of power impossible. We have already seen that contractures that fix the limb in one position and make any movement from this position difficult or impossible, develop readily when the paretic limb is allowed to remain constantly in the one position, and the same holds true for the joint changes. It is consequently essential that the posture of the paralysed limbs should be frequently changed. This may be done by the systematic use of passive movements, but it is more important that the position in which the limbs lie while they are more or less powerless during the first few weeks of the illness should be frequently changed. If you insist that the arm should not be allowed to lie constantly flexed or the leg permanently extended, you will find that there will be much less danger of the limbs becoming fixed in these or in other positions by contractures. Massage is also of considerable benefit, especially when combined with passive movements, but it should be gentle and frequently repeated. Even when contractures have developed massage and passive movement help to overcome them; they can be most satisfactorily employed when the rigidity has been reduced by immersing the limb for a time in a warm bath. When the range of movement is limited at any joint by adhesions in it, these should be broken down by passive movement—under an anæsthetic if necessary.

But we have seen that peripheral stimuli, and especially cold, tend to increase the rigidity of the paretic limbs, and we should consequently attempt to eliminate them as far as possible. These limbs should be kept as warm as possible, and it is often advisable to have the arm rolled up in loose cotton-wool during the early weeks of the illness; this also reduces exposure to other forms of irritation. Finally, as power returns the patient should be urged to move the limbs frequently as much as he can, for unquestionably active movements are the most effective stimulus to the muscles, and practice improves the co-ordination and the range of the movements that return.

June 6th, 1910.

THREE CLINICAL LECTURES
ON
THE HEART.

Delivered at St. Bartholomew's Hospital.

By SAMUEL WEST, M.D., F.R.C.P.,
Physician to the Hospital.

LECTURE III.

GENTLEMEN,—In my last clinical lecture I spoke of cardiac murmurs, and I divided them into two groups—those which were associated with organic disease of the heart, and those which were not. And I said several things about the first group. I now propose to say something about the second group, or those murmurs which are called functional. These murmurs are nearly always systolic, and soft or blowing in character, while the sounds of the heart are present and often not much altered. They may be heard at the base, at the apex, or over the whole præcordium. At the base they are heard on both sides, over the pulmonary orifice on the left side and over the aortic on the right. A great deal is said about murmurs on the left side, but not much about those on the right, and I shall have something to say about both.

Functional murmurs at the pulmonary base are extremely common. Sometimes they are called pulmonary murmurs, but often also hæmic or anæmic murmurs, because so commonly associated with anæmia. Whether they are hæmic in the strict sense of the term, that is to say, due to some altered condition of the blood, as is often held, is open to question. There are many objections to that view. First of all they vary greatly at different times, yet the condition of the blood does not change, and they vary much with position, so that they may be distinct when the patient is standing and disappear when the patient lies down.

The most probable explanation is that which is given for most other murmurs, namely, that there is a dilatation of the vessel above the valve. Whatever may be the case with the other apertures of the heart, the ring to which the pulmonary valves are fixed is rigid and does not alter in shape or size, but the vessels above may, and are very likely to do so, as, indeed, we can see they do in some conditions. So the probabilities are that the usual explanation holds, viz. that the blood is passing out through an ordinary aperture from or into a somewhat dilated vessel—the very condition to produce an eddy, and in consequence a murmur, just as occurs with an aneurysm or permanent dilatation of an artery. Pulmonary murmurs vary very much with the force of the heart, and that may be the reason why they are not heard so distinctly when the patient is lying down. They are often associated with murmurs in the veins of the neck.

Venous murmurs at the root of the neck are generally continuous, often musical, and vary with the rapidity of the blood-stream, so that they are loudest on inspiration, for the blood is then being sucked into the chest, and in the erect position, because then blood has gravity to help it in falling into the chest. These murmurs can be easily proved to be produced in the veins, for a very little pressure on the veins above will cause the murmur to disappear, the murmur returning again so soon as the pressure is removed, the pressure used being too little to have any effect on the arteries. The French name for these venous murmurs is *bruit d'diable*, and a curious noise it may be sometimes. The Germans have a more poetical term, "Nonnengeräusch," the murmuring of nuns. This conveys the idea of the low hum which many of these murmurs have.

Functional murmurs at the aortic base.—There is not much said about these murmurs in books, yet they are very important.

There is no condition in which patients seem to be in a condition of greater nervousness or excitement than when they come to have their chest examined for the purposes of life assurance; possibly much may turn on the result, their marriage, for instance, or a business partnership. The heart is then found beating rapidly, and a soft systolic blowing murmur is often heard at the aortic base. Before condemning such a case as one of organic disease, let the patient sit down, and be examined again later. As soon as the excitement passes the murmur disappears. Many candidates have been very unfairly rejected for life assurance because of such transient murmurs. I remember a gentleman telling me that he presented himself for assurance, when he was a young man and going to get married. The doctor examined him, then with great solemnity, said, "Young man, walk through life," and declined his proposal. He lived,

however, to a good old age, and never had a cardiac symptom or murmur all his life. You must remember that in insurance work you have a duty to perform to the candidates as well as to the office, and your position is that of arbitrator, adviser, or intermediary between the office and the candidate, and if you are unfair to the candidate the office loses business.

That arteries should dilate is not surprising. You can see it in every blush. It is only necessary that the blush should affect a big vessel for a murmur to be produced, and this may easily happen in nervous, excitable patients, men or women. There is a curious condition, which was described by Sir James Paget many years ago, in which dilatations of the arteries occur which are like aneurysms, but are not aneurysms, because they are not permanent; these Paget called **mimic aneurysms**. Sometimes you may see them come and go. I have seen beneath the clavicle a pulsating swelling over which the veins were dilated, the percussion was impaired, and a systolic murmur audible; but after a little while all these signs were gone. This may occur in almost any vessel of the body, and is not rare in the abdominal aorta, in that condition which Laennec described as *abdominal pulsation*. Many such cases have been wrongly diagnosed as aneurysm of the abdominal aorta. The systolic murmur audible over the enlarged thyroid in exophthalmic goitre is usually referred, no doubt rightly, to arterial dilatation.

There is a practical conclusion to be drawn from what I have just said.

It is a common practice when the heart has been examined and nothing wrong found, to make the patient walk about, go upstairs, or do some muscular exercises, and then to listen again. A systolic murmur may then very likely be heard, but that proves nothing. If there be organic disease, a little extra strain might make the murmur due to it more evident, but if a transitory murmur is produced in this way only it is of no value as indicating valvular disease; nor is the administration of nitrite of amyl, which is sometimes given for the same purpose.

Functional murmurs heard at the apex or over the body of the heart.—The peculiarity of these murmurs is that, although they are heard at the apex and over the body of the heart, they are not heard outside the limits of the heart. They are very often called *dilatation-murmurs*, and it is a good name for them, yet it mixes up fact and opinion in a way which I always protest against, although in this case I think the explanation correct, for they are found when dilatation is present, they disappear when the dilatation goes, and they return if the dilatation recurs. So they come and go and vary directly with the dilatation. On the other hand, not all cases of dilatation have murmurs, *i.e.* dilatation may occur without murmurs, but that is another matter.

The commonest of these murmurs is that heard in chlorosis. Here, besides the pulmonary murmur, a systolic murmur is heard over the whole præcordium. The heart may be easily made out to be dilated, and the murmur vanishes when the dilatation passes off, as it readily does after a few hours' rest in bed, to return again if the patient get up too soon and the heart dilate again.

In a case of double aortic disease, where the left ventricle is dilated and hypertrophied, murmurs are often heard at the apex, and it is hard to tell whether they are the murmurs of the base transmitted to the apex or something different. Usually they differ in character entirely from those at the base, and instead of being distinctly systolic and diastolic, form an almost continuous churning. These, again, are dilatation-murmurs, and if the dilatation passes off, change their character or disappear. One variety of this dilatation murmur has been called *Flint's murmur*. This is a diastolic murmur, and was described by Flint as a presystolic murmur not associated with mitral stenosis. I have heard these murmurs frequently. They were familiar long before they were described by Flint. Though diastolic and, it may be, presystolic in time, they are not in the least like the pre-systolic murmur of mitral stenosis, and could not be confused with it.

Some of these left ventricle dilatation-murmurs are loud and musical, and may be heard even at a considerable distance from the body. The most remarkable instance of this kind I have met with occurred in a case of paroxysmal tachycardia, in which the heart became greatly dilated during the attack. The man was in the police force, and was put upon the sick list because his neighbour in the dormitory complained that the man's heart made such a noise that it kept him awake. I think this may have been more or less true, and I have no doubt the murmur was heard by the man who slept next to him some feet away, though I myself never heard it more than twelve to eighteen inches away from the chest. The murmur used to come and go with the attacks. Ultimately the man got quite well and returned to active duty.

These dilatation-murmurs are generally explained as due to regurgitation through the mitral and tricuspid orifice. You will remember how the muscle-fibres of the heart turn over the ring of the mitral and tricuspid valve, and you can imagine that when these muscle-fibres contract they might pinch and contract the ring. Experiments were made to see

if that was true by Ludwig and Hasse. The hearts of animals were cut out of their bodies and at once plunged into strong chromic acid, which caused them to contract. That kind of experiment, one would have thought, was of too violent a nature to justify very sweeping conclusions. Yet on the strength of these it was asserted that the valves of the mitral and tricuspid orifices were not competent to close those orifices completely unless the fibrous rings have been first of all constricted or pinched together by the muscular contraction. Consequently, if the muscles did not contract properly those rings would not be pinched, the valves would not be competent, and regurgitation through the valves would occur. This is now the explanation generally accepted of these so-called dilatation-murmurs which are heard over the left and right ventricles respectively.

It is difficult *à priori* to accept such a theory and to believe that an important organ like the heart has so little reserve that slight weakness of the muscle should lead to such defective action of the valves as to interfere enormously with the action of the organ as a pump. It is not safe to set *à priori* considerations against the results of experiments, and there is another and better line of argument to prove this theory incorrect. All physiological observations and theories must bear the test of application and investigation at the bed-side, and this does not bear that test. The regurgitant mitral murmur due to organic disease of the valve is heard at the apex, and is propagated into the axilla and to the left interscapular space behind; this at any rate is the general rule. If the dilatation-murmurs we are speaking of were due to regurgitation they ought to be heard like the ordinary regurgitant murmurs, and with equal frequency, in the axilla and behind. They are not, and consequently it is begging the question to assume they are produced in the same way. Sometimes when the heart is greatly dilated, these dilatation-murmurs may be heard in the axilla and behind, and post-mortem observations show that the mitral and tricuspid orifices are enormously dilated, so that several fingers may be passed through them. In such cases the murmurs are due to regurgitation no doubt, but this exception really proves the rule, for if some of these murmurs are propagated as regurgitant murmurs should be, and others are not, they cannot all be produced in the same way.

There are good reasons, then, for not accepting that sphincter action of the muscle upon the *mitral orifice*.

The *tricuspid valve* is more easily examined, but organic tricuspid disease is very rare. When tricuspid disease is present the regurgitant murmur is heard over the lower part of the sternum, and propagated distinctly almost horizontally across to the right side. If a systolic murmur be propagated in that way the diagnosis of tricuspid regurgitation is certain. In the same way tricuspid stenosis may be easily recognised. I have diagnosed organic tricuspid disease several times during life, and have verified the diagnosis post-mortem.

Tricuspid disease is very rare, yet the diagnosis of tricuspid regurgitation is very often made. What, in the majority of cases, does that diagnosis rest on? The fact, perhaps, but by no means always, that there is a murmur. But if so, it is a systolic murmur heard locally and not propagated to the right, and therefore not propagated as it should be. But where, as is often the case, there is no murmur at all, the diagnosis rests upon entirely different considerations. It is made on the grounds that there is great dilatation of the right auricle and right ventricle, that the veins are visibly pulsating in the neck, and that there are obvious signs of venous obstruction. But those facts are not of themselves definite proofs of tricuspid regurgitation. In the vast majority of these cases of right-sided dilatation there is no murmur at all, and why is this so if the valves are incompetent, and why, when there is a murmur, is it not propagated as it should be?

So in dilatation of the right ventricle, as of the left, there is very strong clinical evidence against these murmurs being really due to regurgitation. They may be called dilatation-murmurs because they come and go with the dilatation, and this name implies no theory. It merely correlates two facts, the murmur and the dilatation, though it also indicates the opinion that the murmur is associated with the dilatation and not with anything else.

June 6th, 1910.

THE HYPERÆMIC TREATMENT OF INFLAMMATION.

By A. V. POYSER, M.B., B.C.

THE treatment of inflammation by methods inducing hyperæmia is far from new. Inflammation itself is but a symptom of the temporary victory of pathogenic organisms over the body tissues, and the signs of inflammation are chiefly due to an increase of the blood-supply—a protective process designed by Nature to fight bacterial organisms. It has long been recognised as a clinical fact that in mitral disease, particularly with stenosis of the valve, phthisis is a very rare condition. Conversely, with pulmonary stenosis the occurrence of this disease is relatively increased. That is, the hyperæmic condition antagonises the growth of organisms, but their multiplication is favoured by a condition of relative anæmia.

Even the application of this principle as a therapeutic remedy is not new. Instinct leads an animal to suck its wounds, and the same instinct is one of the most popular of home treatments in the human race. The application of hot cloths, fomentations, or poultices is again only another method of drawing more blood to the diseased part. That "nothing is new under the sun" is an old but true quotation. Hippocrates, in paragraph 4 of his treatise, 'The Doctor's Workshop,' described the compression of veins of a diseased part as a means of treatment. The method was used for several centuries, and during this period Galen is said to have added his recommendation. Its use, however, finally fell into disrepute, and lay dormant for many years. But it reappeared, this time in Edinburgh, in the early nineties. It was not, however, until this century that it became recognised as a method of treatment, sound in both theory and practice, and its applications properly defined.

The credit for this lies with Prof. Bier, of Bonn, and it is with his name that the treatment will ever be associated.

Hyperæmia may be of two kinds—active and passive. In the former variety the increased amount of blood is equally distributed between both the arteries and the veins. In passive hyperæmia only the venous circulation participates in the increased flow. Treatment by active hyperæmia has long been one of our safest remedies in the form of massage, counter-irritation, hot fomentations, and similar appliances. Of recent years hot-air baths have produced a more general but similar result. The treatment by passive hyperæmia is, however, the one more intimately associated with the name of Bier. This is usually produced in one of two ways—either by an elastic band causing venous compression, or by dry cupping.

But in what way does the condition of hyperæmia counteract the effects—or rather the causes—of inflammation? It has several effects; some are readily intelligible; others, and perhaps the more important have not, at present, advanced beyond the conjectural stage. The more obvious ones can be termed mechanical. The hyperæmia causes a dilution of the toxins so that they are the more easily overcome by the normal powers of protection of the body. Again, if an opening be present, whether made by an incision or by the bursting of an abscess, the increased exit of serum produced by the hyperæmic condition can be observed to actually wash away the poisonous substances. Coincident with the hyperæmia is an increase in the number of phagocytic leucocytes; the serum is itself relatively increased, which, acting as a nourishment for these white cells, aids them in exerting their protective influence.

But there are other effects of the hyperæmic condition, which are far less easy to understand and as yet are not actually proved. It is well known that the polymorphonuclear leucocytes have the power of destroying bacteria, but it is also granted that other substances are produced in the body to counteract the toxic effects of micro-organisms. One cannot truthfully say that these substances are known. In all probability they are many and various. One class can be grouped together as antitoxins, or stimulins, as Metchnikoff calls them. These are albuminoid substances present in the blood, which are stimulated into activity by the circulating toxins of the bacteria. They act directly upon the leucocytes, energising them to perform phagocytosis. In another class are the opsonins of Prof. Wright; they are bodies produced in the muscle-tissues, which act, not upon the leucocytes, but upon the bacteria, causing them to be more "prepared for being eaten" by the leucocytes. But the opsonin present is not specific for the invading organism. It possibly exists as an "opsinogen," which, meeting the particular bacteria, is then converted into the opsonin specific against that organism. The presence of such substances has now been demonstrated by Wright. Metchnikoff, however, gives them quite a secondary place to phagocystosis, considering that if they are of value it is only as an aid to phagocytosis. Whatever may be the nature or composition of these substances, and wherever they may be produced, to be of value they probably at one time must enter the blood-stream, and it may be owing to their presence there that Bier's method of hyperæmic treatment is so valuable. That this treatment is not merely conjectural is proved by the fact that after a period of passive hyperæmia the appetite of the phagocytes for bacteria—or opsonic index—is increased. This has now passed the conjectural stage. Opsonic indices taken in tuberculous joints, after treatment by passive hyperæmia, have

been found to be always increased. In diabetic subjects the opsonic index for *Staphylococcus aureus* has been found to vary between ·32 and ·72—that is, the index is uniformly low. This illustrates the excessive liability of diabetics to boils, carbuncles, etc., and the rise in the opsonic index after treatment demonstrates the beneficial results of the hyperæmia. Again, if there are multiple inflammatory foci present in the body, and one of them is subjected to Bier's treatment, it is found that improvement occurs in all. This can arise only from the production of immunity-producing bodies at the site treated and by their being afterwards disseminated all over the body by the blood-stream. Summing up, it can be said that the production of hyperæmia is an artificial stimulation of the dormant anti-microbic processes always present in the body.

The application of this treatment is as wide as inflammation itself. It can be used for inflammation, whether acute or chronic, and in almost any situation in the body. At first its use was practically confined to the limbs, their shape and position allowing congestion to be easily produced. Its field recently has enormously increased; by ingenious applications, such remote parts as the lungs or the female pelvis can now be brought under treatment. It has been mentioned that there are two kinds of hyperæmia—active and passive—and that the passive variety is produced almost entirely by either constriction or by "cupping."

I will first describe the production of passive hyperæmia by means of constriction. This method is confined to those parts where pressure can be applied so as to produce hyperæmia distally to the constricting apparatus. It is best produced by an elastic band, such as elastic webbing, an Esmarch's or a Martin's bandage. The last is perhaps the best; it is easier to keep clean, and is not so rigid as the Esmarch. It must be placed around healthy tissues, and if possible some distance away from the infected focus; thus if the wrist is being treated the band should preferably be above the elbow; if the foot is the affected part the constriction should be placed above the knee. It is advisable to place some soft material—boracic lint is good—between the elastic and the skin, and the site of constriction should, if possible, be altered at every time of application. Bier recommends that from six to eight turns should be made, each overlapping the preceding one by half an inch. Only venous congestion should be produced, and for this the turns must not be made too tight—the fingers should be able to pass easily under the elastic band. With the correct amount of pressure the limb swells and becomes slightly cyanotic; a definite blue colour should not be produced, however, except near the termination of the period of constriction. The arteries should on no account be subjected to pressure; if this happens the limb becomes pale and the distal pulse impalpable. Neither should any pain be produced; pain previously present ought to disappear or to be relieved after about fifteen minutes' application of the bandage. Pain produced by inflammation is due to an irritation of the nerve-endings by the toxins, and it has been suggested that the alleviation of the pain is due to the dilution of these toxins. According to Bier himself, "the degree of obstructive hyperæmia is a correct one if the patient is not in the least distressed by the presence of the bandage." Very often, however, pain is complained of by patients when, as far as one can judge, the bandage is not too tightly applied, and when good results, indicative of good methods, are being obtained. To explain this someone has suggested that Bier's statement applies only to the Germans, and that the English, being somewhat less phlegmatic, are more readily affected by pain. Some knowledge of the degree of obstruction can be obtained by a careful observation of the size and tension of the superficial veins, and after some experience this forms a useful guide to the correct amount of constriction. It is very essential that complete venous stasis should not occur—the circulation of the veins must only be impeded; to prevent this undesirable occurrence the circulation in the veins should be observed by watching the re-entrance of blood after temporarily emptying them. This can be done with the hand by stroking away the blood in the superficial veins. When it is seen that the correct degree of constriction has been obtained, the marking of the turns of the bandage facilitates the next time's application. The length of time the bandage is left on varies considerably with different practitioners: the more acute the condition the longer should the constriction be maintained. The maximum length of time for an acute case should be ten hours; the bandage should then be left off for two hours. It

should never be reapplied until the congestion and œdema produced by the previous constriction have disappeared. In chronic cases two to six hours a day is a sufficient length of time, and the bandage should be a trifle looser, so that a definite blue colour in the limb is never produced. In practice, however, the period of time during which the bandage is kept on does not appear to be all important.

In my own cases, some of which are quoted later, the constriction was usually maintained for two hours, and then removed for a like period. During the night it was taken off altogether. On the whole the results were exceedingly good.

If there is pus formation the abscess must, of course, be incised, but here, again, Bier's treatment is valuable. The incision need only be of sufficient size to allow the escape of the pus. In Bier's words—"The knife deals with the pus, the hyperæmic treatment fights the bacterial invasion."

There is no reason why this treatment should not be combined with others; indeed, excellent results have been obtained in tuberculous joints by the use of Bier's treatment with a previous injection of iodoform emulsion. In acute cases the simultaneous application of hot fomentations assists in the production of hyperæmia with its resulting beneficial effects.

A passive hyperæmia produced by cupping, or suction as it is better termed, is identical with that following constriction. Cupping has the advantage that it can be applied in situations where an elastic bandage cannot be placed, *e.g.* on the trunk, and it is certainly far more convenient for the neck or head.

The cupping apparatus is now made in every possible shape to fit almost any part of the body. It is in the form of the old dry-cupping glass, exhaustion being produced by means of an india-rubber ball and tube.

It is applied for half an hour or an hour once or twice daily. During, say, one of these hour periods, it is put on for five minutes and then removed for five minutes; the suction therefore is really exerted for only half the time.

The cup should be of such a size and shape that it comes in contact only with healthy skin.

As the period is so much shorter than when constriction is used, the power exerted is relatively somewhat stronger. A moderately sized dome should be produced. Its colour should be of a reddish-blue; pallor must here, again, be avoided, as this denotes anæmia. The pain is not relieved to such an extent as in the former method, but the suction must be such that it is easily tolerated.

Suction as a method of treatment is also exemplified by the use of Kuhn's mask. This is placed over the respiratory orifices, a negative pressure is produced, inspiration is obstructed, but expiration unimpaired. A passive hyperæmia is thus created and tuberculosis of the lung directly attacked at the actual site of the disease.

Active hyperæmia means that there is a general congestion of the part, the arteries as well as the veins being affected. Hot fomentations, poultices, etc., are an old-established means of procuring this condition. Massage—at any rate of an organised character—is of more recent date.

Hot-air baths are of still later introduction. They rival suction glasses in their many shapes and sizes. They are now adapted to fit almost any region of the body, and are much used, giving good results in the treatment of intra-pelvic inflammations and other equally inaccessible regions.

Simple forms of apparatus are easily manufactured out of a bed cradle and blankets with a funnel, tube, and spirit lamp. Of whatever character the chamber is, the temperature should be kept at about 240° F.; a temperature below 200° is of little use; one of 300° or over cannot be borne by the patient.

During the first applications fifteen minutes is a sufficient length of time; later, this can be gradually raised to thirty to forty minutes. Hot-air douches is another method of producing an active arterial hyperæmia. This method used *per vaginam* is applicable to intra-pelvic inflammations.

As has been stated, practically all inflammations can be made to react to treatment by hyperæmia. The principle in all cases is the same; the form of treatment largely depends upon the situation of the inflammatory condition. In acute superficial inflammations, such as erysipelas and lymphangitis, either the bandage or suction acts equally well. Within twenty-four hours or so a diminution of the acute condition becomes marked, and the general symptoms rapidly clear up. The results in cellulitis are equally noticeable. *Acute mastitis* reacts wonderfully well to this form of treatment; if the breast is pendulous the elastic bandage may be used, but a suction glass is usually indicated.

Whitlows, owing to the easy application of the constriction, have been more universally treated by passive hyperæmia than almost any other inflammatory condition. The bandage is placed round the root of the finger; the force exerted should be such that the reddish tinge is never lost.

Boils and *carbuncles*, commonly situated at the back of the neck, are best treated by the cupping glass, and react well. If the cupping glass is not obtainable an efficient hyperæmia can be produced by the application of an elastic band placed as low down on the neck as possible. All forms of abscess, including suppurating glands, after an exit has been made for the pus, give excellent results with hyperæmic treatment.

In chronic inflammations tuberculous joints have probably received more attention from this method of treatment than any other condition. The results have not been universally good, but many of the failures may possibly be due to an improper application of the treatment and lack of patience. Perhaps the best results have been obtained by the previous injection of iodoform emulsion. Chronic septic bone conditions, including lack of union in fractures, also clear up quickly after a course of hyperæmia. Sinuses of all sorts, particularly those following operations for empyema, and to a less extent for appendicitis, often resist the common methods of treatment. The application of a suction glass, in the manner detailed above, for one hour daily, causes a profuse discharge of pus and serum, and the sinus usually closes with rapidity.

The treatment of stiff joints, in which it is eventually possible to obtain movement, is now usually carried out by massage and passive movements. If these methods are also combined with hyperæmia movement is more quickly obtained. The ligaments and connective tissue become softened by the increase of serum necessarily produced in the joint, thus giving an increased advantage to the treatment by massage, etc. Chronic skin diseases, particularly such conditions as œdematous eczemas and callous ulcers with large discharging surfaces, give good results with the bandage or suction-glass. In both conditions a copious discharge of serum occurs, and the affected area becomes quickly covered by skin.

It has been found that there is an increased rapidity of cure in treating lupus when the Finsen rays are combined with hyperæmic treatment.

In gynæcological conditions the treatment is also useful, but here a state of passive hyperæmia is difficult to obtain; hot-air baths surrounding the pelvis bring about, however, the allied condition of active hyperæmia. The diseased deep structures partake equally with the superficial ones in the increased blood-flow. Chronic exudative processes occurring within the pelvis, whether following confinement or operation, are those conditions which respond best, but various menstrual disorders have greatly benefited. In perimetritis or parametritis operative treatment is difficult, and is generally to be avoided. Hot vaginal douches and the application of various tampons, combined with medical treatment, sometimes cure, but more often cause only an alleviation of the symptoms. A rapid disappearance of the exudate usually occurs after hyperæmic treatment.

Such treatment is only contra-indicated if the inflammatory process is still acute.

It has been stated above that practically all situations can now be reached by the treatment of hyperæmia. The contents of the thoracic and abdominal cavities are, of course, out of reach except by the use of hot-air baths; there is also one other situation—the hip—where treatment by passive hyperæmia has not yet been made applicable.

The head and neck are within our reach; an elastic band around the lowest part of the neck can be worn without inconvenience. Discomfort from pressure on the trachea can be obviated by pads placed on each side; tapes passing under the axillæ anchor down the bandage and prevent its "riding up." In the shoulder, venous obstruction can be obtained by compressing either the subclavian or axillary veins by means of pads, which are easily maintained in position by tapes passing under both axillæ. Anterior urethritis or inflammatory conditions of the penis can be attacked, preferably by india-rubber tubing placed around the root of the organ. The scrotum can be treated in a similar way; and lastly, suction can be advantageously employed for inflammatory conditions of the posterior urethra.

The following are examples of a few cases treated by me, under Mr. Wallis's guidance, in his beds at Charing Cross Hospital:

CASE 1.—Stableman, æt. 60 years. On July 31st, 1908, he was "savaged" by a horse. When

he first came up to the hospital he had a compound fracture of the lower third of the ulna, the ends of the bone being easily felt through a lacerated wound on the inner side of the arm. There were several other ragged wounds on the back of the arm. He was admitted on the following day. The next day the whole of the lower forearm was very inflamed and stinking.

Hot fomentations and a continuous boracic bath were ordered. Perchloride (1 in 8000) was afterwards substituted for the boracic. The temperature was raised to 101° F., and deep suppuration occurred. In a few days, however, the acuteness subsided and the temperature came down to normal. The condition now became subacute and continued so for over four weeks; several abscesses formed and had to be opened. At this time Bier's treatment by means of an elastic band above the elbow, one hour on and one off, was instituted. There was an increased amount of discharge and much slough came away.

Sixteen days later all inflammation subsided and he was discharged. He continued to attend as an out-patient, and a month afterwards there was firm union, with good, though limited, movement.

Case 2.—Female, æt. 47 years. Admitted December 18th, 1908, with lymphangitis of right foot and leg. Temperature 103·8° F., and general malaise. The condition was a typical severe lymphangitis. There was œdema of the foot and enlargement of the inguinal glands. The condition followed the removal of a suppurating corn. She was ordered hot fomentations and Bier's treatment, one hour on and one off; the elastic band was placed above the knee. On the third day the temperature, after gradually falling, reached normal. Three days later all the redness had disappeared, and she was discharged ten days after admission.

Case 3.—Boy, æt. 6 years. On December 18th, 1908, he was knocked down by a cart, which passed over the outstretched hand.

The skin was divided over the ulnar margin of the hand and in all the clefts of the fingers. The skin of the dorsum was completely separated from the underlying structures. The metacarpal bones of the thumb and middle finger were fractured. The hand and cuts were enveloped in dirt.

An attempt was made to cleanse the hand and sew up the cuts. He was then admitted. Suppuration occurred three days later, with a temperature of 100·5° F. He was put on Bier's treatment, two hours on and two off. Gangrene occurred in the skin over the dorsum of the hand. On December 20th the temperature was normal and the granulations healthy, so Bier's treatment was stopped and dry dressings applied. For the three following nights the temperature rose above 99° F., and so a return was made to the previous treatment. The temperature became normal again within two days.

Bier's treatment was finally discontinued on January 12th. Skin-grafting was attempted, and the boy was discharged on February 26th, 1909.

During the whole time he was instructed to move his fingers as much as possible, particular attention being paid to opposing the thumb and the fingers. Movement in the fingers when the patient was discharged was fair, but the hand as a whole was severely crippled.

Case 4.—Labourer, æt. 43 years. On January 18th, 1909, he came to hospital with a compound "Colles'" fracture, caused by falling off a step-ladder.

He attended as an out-patient very irregularly, and on February 12th had to be admitted. The hand was then very much swollen and inflamed; there was no union; he was very ill, with a temperature of 103·4° F. On the following day he was put on perchloride baths and Bier's treatment, applied above the elbow, one hour on and one off. By the 16th the temperature had gradually fallen to 99° F., and on the following day the temperature was normal.

Pus and serum were discharged copiously. On the 26th only dry dressing was necessary. He attended as an out-patient for some time. Union became complete and very fair movement was obtained.

Case 5.—Boy, æt. 4 years. He was admitted with tuberculous disease of the astragalus, with abscesses. These burst when admitted. Bier's treatment was immediately started, and caused slight improvement of the condition. He was then sent to a convalescent home, the Bier's treatment ceasing. He apparently had only slight attention when away, and returned when amputation was necessary.

Case 6.—Lift-boy, æt. 16 years. Admitted May 27th, 1908, with a compound fracture of the femur and a fractured patella from a fall down the lift-shaft. Both bones were wired, the patella doing well. Suppuration occurred around the ends of the fractured femur. Two months later the suppuration was chronic in character, india-rubber drainage-tubes passing right through the leg were necessary, and there was no union.

Two months later, after several incisions and an operation for sequestrotomy, there was still no union, and the suppuration was unchanged.

Bier's treatment was then started. In six weeks' time, in spite of some suppuration being still present, union was obtained, and owing to his anæmic condition he was quickly got up on crutches. A fortnight later he walked a few steps without assistance, and was discharged with a useful limb.

June 6th, 1910.

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CONTENTS.

PAGE

*A Clinical Lecture on a Case of Syphilis. By W. Hale White, M.D., F.R.C.P. 145

*Treatment in Septic Conditions. By Edred M. Corner, B.Sc., M.B., M.C., F.R.C.S. 151

*Two Clinical Lectures on Hemiplegia and its Treatment. By Gordon Holmes, M.D. Lecture II 156

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A CLINICAL LECTURE

ON

A CASE OF SYPHILIS.

Delivered at Guy's Hospital.

By W. HALE WHITE,

Senior Physician and Lecturer on Medicine to the Hospital.

Gentlemen,—This afternoon I want to discuss with you the case of the man whom you see before you. He is a sailor, æt. 40 years. He came into the hospital about a month ago, complaining of general wasting and pain in the legs, and a painful swelling in the arm. He said he has had malaria; he has been all over the world. As far as he knows he has not had syphilis, but he admits he may have had it. Six months ago he began to feel ill, and he has lost three stone in the last year. Six weeks ago a swelling in the right arm appeared; it has gradually been increasing in size, and in painfulness. He suffered from pain in his legs, which was worse at night. When we came to examine him we found that he was considerably wasted, and that he had a soft swelling of the lower part of the right humerus on the inner side; the swelling is attached to the bone; you will see that the skin is freely movable over it. It extends three inches up the arm, and then shades off gradually into the normal thickness of the arm. It is not connected with the elbow-joint, which is normal; it is somewhat tender, it is not indurated, it is not red, it does not infiltrate the skin. There are one or two slightly enlarged glands in the axilla on the opposite side, and one or two in the groin. Further, we found on each of the thighs a similar swelling connected with the femora. Higher up on the inner side of the right thigh there is another similar swelling. As you can see, these swellings are diffuse, that is to say, they shade off into the

normal tissues. Over none of them is the skin adherent, but they all are painful. On examining the man still further we found that his liver is enlarged. On admission the liver reached to the umbilicus, and at the present time it reaches down below the ribs. It is uniformly enlarged, but nobbly in parts.

Now, the problem was, What was the matter with this man? And I think the best way to take the case will be to consider what could possibly be the cause of the enlargement of the lower end of the right humerus, the enlargement of the lower end of both femora, and the enlargement of the inner side of the right femur, for all these swellings were obviously attached to bones. The first suggestion made, because the man had wasted so much, was that he had malignant disease of his bones. But that we dismissed, and we dismissed it for several reasons: in the first place if he had had malignant disease of his bones to such an extent as to have four large bones affected, he would hardly have been as well in general health as he is now. It is true he was a good deal wasted, yet his general health was good in spite of it. In the next place, those swellings were not as painful as they would have been had they been due to malignant disease. Then, again, the skin over them was not adherent to them, as it might have been in malignant disease. There is another thing, and that is, I think, by the time a man has malignant disease of the liver and three bones affected, the disease would hardly stop at three bones. My experience has been that when we have several bones affected in the body in malignant disease we have a great many affected; it is the rule in such a case to find many outcrops of tumours in various bones of the body. For these reasons and the fact that the man had no secondary deposits elsewhere we came to the conclusion that the man had not malignant disease of his bones. It passed through our minds whether possibly he had tubercular disease of them, but in tubercular disease of the bones I think by the time you have four large tubercular tumours of bones some of them would be softening and breaking down, the skin would have been adherent, and there would have been what the old surgeons called cold abscesses in connection with them. But there was nothing of that sort in connection with this man. Then we came to the conclusion that we ought to consider syphilis. And we decided that these tumours in this man were syphilitic. And the reasons we came to this conclusion were these; in the first place, the tumours are rounded and shaded off indistinctly, they are not localised tumours of bones with sharply defined boundaries. In the next place, they are not particularly tender, and the skin over them is not implicated. They are multiple; there is a good deal of thickening of the periosteum around the bone, as you can tell by feeling the tumours. So for these reasons we concluded that he had four large nodes of his bones. And that they were caused by syphilis was supported by the fact that he complained of a good deal of pain, which pain was worst at night. The next thing was to get him X-rayed. If these nodes had been malignant tumours we should have expected a local tumour of the bone itself. Röntgen rays were used, and here are several pictures showing the results. Here is a picture of the lower end of the humerus: there is not only considerable osteitis and thickening of the periosteum but there is some swelling of the soft tissues all round, and that shows you very well the sort of thing you would expect in syphilis. Here is a skiagram of the femur, and that shows better still a point which is very characteristic of syphilitic disease of bone—that the periosteum is much thickened. Here is a picture of the other femur, and the two together show what is so characteristic of syphilis, that the periosteum is affected all round the bone. Here is another skiagram showing the same thing—thickening of the periosteum, increased density of the bone because of the osteitis, and thickening of tissues round the lower end of the femur. These pictures support the view that he is not suffering from either tubercle or new growth, but from syphilitic inflammation of the periosteum of the bone. Here are some pictures from other cases of syphilitic disease of long bones which Dr. Jordan kindly lent me. They also show thickening of the bone and of the periosteum. Here is one in which the thickening of the bone was so considerable that it was cut down upon, but it was found to be due to syphilis. Injury often determines the existence of syphilitic nodes, and that is why they often occur on the tibia, because the tibia is a bone which is very liable to get knocked. This man complains not only of swellings of his bones, but also of considerable wasting, and please remember this lecture for this if for nothing else, that wasting is not uncommon in

syphilis. When you see a patient who is wasted say to yourself: Has he new growth? Has he tubercle? Has he syphilis? Since the treatment, which we will speak of directly, the man has gained in weight, though he has had no special diet, from which I think we may conclude that the wasting was one of the evidences that the man had syphilis.

The only thing which we have not yet discussed about this man's case is the condition of his liver. Some of you objected at the time he was first seen that the hepatic enlargement did not fit in with the diagnosis that the patient was suffering from syphilis, because the liver was rather uniformly enlarged. It was uniformly enlarged, I admit, but I do think—and I examined it several times—that it was lumpy; there seemed to be one lump under the upper part of the rectus, and another further out to the right. So that raises the question, What can syphilis do to the liver? Very rarely, in the early stages of syphilis, patients get jaundiced, but this jaundice soon passes off. What that is due to is not known, but it is so rare that it really hardly enters into clinical medicine. So putting that on one side we come to the tertiary symptoms of syphilis as it affects the liver. Syphilis can do three things to the liver: it can produce an increase of fibrous tissue in the liver, it can produce gummata in the liver, and it can cause lardaceous disease of that organ. Take the last first. Lardaceous disease is not nearly so common now as it was thirty or forty years ago, and that is chiefly because we do not have, owing to the advances in surgery, wounds left to suppurate for the long time that they had to be left many years ago. So that the most important cause of lardaceous disease no longer exists. Further, syphilis is now much better treated than it was half a century ago, consequently the lardaceous disease following syphilis is not so often seen as it was half a century ago. But we did not think this man was suffering from lardaceous disease because the liver was irregularly enlarged, and because he had no albumen in his urine indicating lardaceous disease of the kidneys. But a point in favour of lardaceous disease was that the spleen was enlarged somewhat. On the other hand the spleen was not nearly as large as we should have expected it to have been had it been affected with lardaceous disease. So we considered that the balance of evidence showed that this man had not lardaceous disease of his liver secondary to his syphilis. We were therefore forced to conclude that other results which follow syphilis were the causes of the enlargement of the liver. And you will remember I told you that the other things which syphilis can do to the liver are to produce fibrosis and to produce gummata. I went through our post-mortem records here and I found that in twenty years 95 instances of syphilis of the liver (excluding lardaceous disease) were found in the post-mortem room. During that time 9500 post-mortems were performed. So you see that about 1 per cent. of all the people who die in a general hospital show evidence of syphilis of the liver by the presence in it of fibrous tissue or gummata. Congenital syphilis can produce the same effects, but deaths in people who have congenital syphilitic disease of the liver are so rare that all the 95 cases were instances of acquired syphilis. The exact proportion between gummata and fibrous change was, that 23 of the 95 showed gummata, and 72 showed only extensive fibrosis of the liver. Some of the twenty-three had fibrous tissue in the liver also. This subject is of great interest to us at Guy's, because the recognition of the fact that syphilis could affect the liver was largely due to the labours of Sir Samuel Wilks. In 1863 in the 'Guy's Hospital Reports' Sir Samuel Wilks wrote a paper on visceral syphilis, which has become classical. Before he and others made these discoveries all gummata of the liver were thought to be cancers. The fibrous tissue produced in the liver by syphilis is unlike the fibrous tissue which you see in alcoholic cirrhosis, because instead of being uniformly distributed throughout the whole of the liver, the fibrous tissue occurs as great bands running through the liver. Often the bands are one third of an inch wide, and when you cut sections of the liver you see shiny white bands running right through it, cutting up the liver-substance. These shiny white bands often radiate from a single point, so you see, on the surface, depressions, with healthy liver-substance between them. When these are very extreme the liver has even been compared to a bunch of grapes. It is necessary for you to remember that the hepatic tissue between these bands is healthy, whereas in alcoholic cirrhosis of the liver the liver substance between the obvious strands of new-formed fibrous tissue is often itself affected by a finer cirrhosis. Here I show you a specimen of gummata and fibrous

bands in the liver, and you see that the two make a very lumpy liver indeed. As the gummata get absorbed, depressions occur, and thus the liver becomes still more irregular in shape. And sometimes you will see at the post-mortem when these strands of fibrous tissue are cutting the whole liver up like this, that they proceed from the site of a healed gumma. Those, then, are the changes that syphilis can produce in the liver. Hepatic syphilis occurs more commonly in men than in women, in the proportion of about three men to every woman, and the age at which it occurs is, as you would expect, from twenty-five to fifty. Remember that it gives rise to no symptoms, except that sufferers from it may complain of a fulness due to the enlargement of the liver. The condition of the liver which I have now described to you never produces jaundice, never produces ascites, never produces, as far as I know, any symptoms except such as are due to merely the mechanical enlargement of the liver. That point is of great importance, for so many people come before us who have had syphilis and have also drunk immoderately, that you will be in doubt in many from the enlargement of the liver only whether the man is suffering from alcoholic cirrhosis or from syphilis. You may not be able during life to feel the lumps on the liver which I have been describing. If he has jaundice, or if he has ascites, or if he has signs of backward portal pressure, as shown by hæmatemesis, for instance, it means he is afflicted with alcoholic cirrhosis, and is not an example of syphilis of the liver. This man was not jaundiced, and he had not ascites; he had no signs of portal obstruction at all. He had a large lumpy liver, and therefore we concluded he had syphilis of the liver like the specimens which I have shown you. He has been put upon large doses of iodide of potassium; the liver has shrunk very considerably while he has been under observation, and therefore we conclude that although no doubt there was some syphilitic cirrhosis, much of the enlargement was due to gummatous deposits in the liver.

Let us take this opportunity of running over rapidly the other ways in which syphilis may affect the viscera. Let us finish speaking about the abdomen. For all practical purposes, putting aside lardaceous disease, there is no syphilitic disease of the stomach or of the intestines. A small gumma has been seen in the stomach, but it occurs so rarely that it really does not come into consideration in clinical medicine. There is no syphilitic affection of the alimentary tract until you get right down to the rectum, where syphilis does cause fibrous stricture. But that condition is much more rare than is frequently supposed. And it is almost a surgical condition, so we need not stop to consider it further here. During the secondary stage syphilitics may get an acute nephritis, which improves very much on anti-syphilitic treatment, showing that it is probably syphilitic, but this condition is not common. Proof is wanting that syphilis will cause granular interstitial nephritis such as we commonly see, say, in patients who have died of cerebral hæmorrhage. Gummata of the kidney are very rare, and so are gummata of the spleen. Possibly this man has one—at any rate the spleen has become a little smaller under treatment. To finish with the abdomen, you know you may get small gummata in the epididymis. Syphilitic testicle is a comparatively common condition. Two syphilitic changes occur in the testicle, a great increase in the amount of fibrous tissue and a fairly extensive gummatous infiltration. The result is that the testicle is very much enlarged. It may be much bigger than a man's fist, and as the gummatous material is absorbed the organ becomes filled chiefly with fibrous tissue, and ultimately there is a small atrophic testicle. You know the common way adopted in the post-mortem room of trying to find out if a patient has had syphilis of the testicle is to cut it in half, then with a pair of forceps an attempt is made to draw out the tubules, and if there has been much fibrosis and destruction of tubules they will not draw out, and we infer that the patient has had syphilis.

That concludes what I have to say about the abdomen. And now with regard to the thorax. Syphilis will produce laryngitis. The characteristic feature is that the ulceration which follows distorts the larynx very much, so that in a severe case you lose all idea of the relationship of the parts, and there may be some gummatous infiltration of the larynx itself as well. Sir Samuel Wilks pointed out in 1863 that syphilis may affect the trachea. He gives in his article an account of a case in which the trachea was, by syphilitic constriction following ulceration, narrowed down to half its ordinary calibre. Syphilis may in the same way

affect one of the bronchi, and that leads to great difficulties in diagnosis. The patient complains of dyspnœa. You examine the chest, and you find that air is entering badly on one side, and you accordingly wonder whether aneurysm or growth is pressing on one of the bronchi, or whether he has cicatrisation of the bronchus from some syphilitic ulceration. I have known patients needlessly tracheotomised, the trouble being thought to be due to the cicatrisation in the larynx, when really it occurred in the trachea below the tracheotomy wound, and therefore the tracheotomy did not help the patient. But tracheotomy is often useful in syphilitic laryngitis, because, owing to the constant movement of the vocal cords, the syphilitic laryngitis does not have a chance to heal under the anti-syphilitic remedies which are given; it is not arrested until the cords are put at rest, and this is brought about by a tracheotomy. Most cases of bilateral paralysis of the abductors of the cords are instances of tabes, and tabes itself is due to syphilis. Passing further down, we come to syphilitic disease of the lungs. Here are some specimens. Here is diffuse syphilitic fibrosis of the lungs, and here is gumma of the lung. But though I am able to show you these, syphilis of the lung is of little clinical importance at all, it is so rare. I do not think any person can live long enough to have sufficient experience to diagnose syphilis of the lung. Fibrosis is excessively rare; gummatous deposits are almost as rare. Patients who have had syphilis are more liable than others to become the subjects of tubercular affection; therefore, if you discover in a patient with syphilis that there are physical signs in the lungs, the probability is that you are dealing with a case of phthisis in a syphilitic subject, and not with genuine primary syphilis of the lungs. It is important to remember what I said as to syphilis predisposing to tubercle, because the combination of the two diseases enormously increases the gravity of the outlook from the point of view of the tubercle. If a person has syphilis and develops tuberculosis, the outlook is very grave indeed, and especially if he develops tuberculosis of the lungs. Syphilitic disease of the heart is quite exceptional, but you may meet with gummata, and also with fibrosis of the heart. Here I show you an example of syphilitic fibrosis of the heart. You see it best in the intra-ventricular septum, as you always do. Dr. Fagge, in 1874, collected twenty-seven cases of fibrotic disease of the heart, of which several were syphilitic. And then it is interesting to bear in mind that people with fibroid disease of the heart are liable to drop dead without any warning, so that syphilis may be a cause of sudden death. I made a post-mortem myself, when I was doing post-mortems, on an elderly man who was seen to fall down in the street. He was brought to the hospital and was dead on admission. He had well-marked fibroid disease of his heart, and abundant evidence of syphilis. I collected nine cases of cardiac fibrosis. Four were brought into the hospital dead, two fell dead in the out-patient room, and the remaining three were not regarded as seriously ill until death took place. So you see fibroid disease of the heart which may be due to syphilis frequently ends in sudden death. You will find further figures in the Pathological Society's 'Transactions,' showing how liable these people are to die suddenly. Syphilis may cause atheroma of the coronary arteries. Atheroma affecting the aortic valves, when the mitral valve is healthy, is often due to syphilis, and if it occurs in a woman is almost conclusive evidence that the woman has suffered from syphilis. Because of the atheroma that it leads to, you know that syphilis may be the cause of aneurysm. If a woman has an aneurysm it is almost proof that she has had syphilis, because the other main cause of aneurysm of the aorta is strain, and severe strain does not usually take place in the case of women.

So much for syphilis as it affects the thorax. It would necessitate a series of lectures to give you the results of syphilis as it affects the nervous system. As you know, syphilis will produce general paralysis of the insane, will produce tabes dorsalis, and will produce syphilitic endarteritis leading to cerebral softening, will cause gummata of the brain, and paraplegia due to disease of the cord, and therefore you see syphilis will cause many diseases of the central nervous system. It has been computed that in this country about 7000 people die every year from the effects of syphilis in its later stages, and of these by far the greater number suffer from disease of the central nervous system. You will have noticed in what I have said that although syphilis affects the liver very strikingly and characteristically, it does not affect the abdominal organs in such a way as to kill.

In the remaining few minutes I will talk over with you what we propose to do with this man. Although he has been liable to infection with syphilis, he is not aware that he has ever had syphilis, and therefore he has not been treated for it. So it will be necessary for us to do so. When he came in we at once gave him large doses of iodide of potassium. You can give very large doses. For instance you may to begin with give 30 gr. three times a day. I have had patients taking as much as 100 gr. three times a day, that is to say 300 gr. a day. One man I remember took 600 gr. a day. Usually these large doses are given to cases in which there is probably gummatous deposit in the central nervous system, for some parts are so vital that it is essential to get the patient under the influence of the iodide as quickly as possible. To prevent iodism give the medicine well diluted, either in water or in milk. This man has taken it so and has had no iodism. The liver has become smaller under this treatment; on admission the lower border was at the umbilicus, now it is midway between the umbilicus and the ribs, and the swellings on his bones are much less prominent. The question remaining to be discussed is whether he ought to take mercury. I think he ought, because we have no evidence that he has undergone a mercurial course. For most cases probably the best way to give mercury is to inject it intra-muscularly. The reasons are these: in the first place we may be sure that the patient has his dose under that method, because he cannot inject it himself; the doctor must do it for him. To show you how careless patients are about taking medicine by the mouth, I remember seeing a man who obviously had a gumma of the brain and syphilitic retinitis, and when I looked at his eyes I said, "Have you been under treatment?" "Oh, yes," he said, "the doctor gave me medicine to take, and I have been under his treatment two years." Knowing the doctor, it seemed to me so improbable that the disease had not been recognised that I said at once to the patient, "You may have been ordered the medicine, but you have not taken it." "Well," he said, "sometimes I forget it for two or three days together." And that is a common sort of case, that the medicine is ordered to be taken by the mouth, but it is not taken regularly. The great advantage of giving mercury intra-muscularly is that you can be sure that the patient gets it. It is now just twenty years since I began to give intra-muscular injections of mercury. I usually inject with strict antiseptic precautions $\frac{1}{8}$ gr. of perchloride of mercury disolved in ℥vij of water, every evening, deeply into the gluteal muscles, in such a situation that the patient is not likely to lie upon the spot where the injection had taken place. I have been responsible for giving many hundreds of these injections, and I have never seen the slightest harm follow. But it is a distinct disadvantage that it has to be done once a day, for out-patients cannot attend daily. So recently people have taken to injecting metallic mercury, for it is found you can cure the patient even if you do not inject it oftener than once a week. A grain of mercury once a week into the gluteal region is a sufficient dose. There are two chief prescriptions, for both of which we are indebted to Colonel Lambkin, who has done so much in the treatment of syphilis. In one, mercury is mixed with lanolin and carbolised liquid paraffin, and in the other with creasote, camphoric acid, and palmitin. These greasy emulsions of mercury can be injected in just the same way as the perchloride. Some have injected calomel, half a grain once a week. The routine at the Royal Army Medical Hospital is this: Six weekly injections (4 of calomel, 2 of mercury) and then there are eight weeks' rest. Then eight weeks' treatment (4 mercury injections in the eight weeks), and then sixteen weeks' rest; then eight weeks' treatment (4 mercury injections once every two weeks), and then twenty-four weeks' rest. Then sixteen weeks' treatment (4 mercury injections, one every four weeks), and then four weeks' rest; then eight weeks' treatment (4 mercury injections, one every two weeks). That is, two years' treatment by mercury.

A very useful way of giving mercury is to rub it in. That is the method usually carried out at Aachen, where patients go usually for six or eight weeks' treatment, repeated two or three times. The waters there are warm and contain sulphide of sodium, sulphuretted hydrogen, sodium chloride, and sodium bicarbonate. The patient gets up early and walks half a mile to the springs, and drinks a glass of the water, comes back, has breakfast, and rests for half an hour and takes a bath of the natural waters at a temperature of 86° to 90°. After his bath he is rubbed for twenty-five minutes with a preparation of mercury that very closely resembles our ordinary mercurial ointment.

Seventy-five grains of this are used for each inunction. The object of the inunction is to get the mercury rubbed in thoroughly. The rubber does not wear any glove on his hand. The mercury is rubbed the first day into one thigh, on the next day into the other thigh, then into one arm and then into the other, then one calf and then the other, and so on on alternating parts, so that at the end of a week the rubber can begin over again. It does not matter much where you rub it in so long as it is not into the pubes or the axilla; they are very hairy, and you may set up dermatitis if you do that, but the back is an excellent place.

The third way of treating syphilis is to give the drug by the mouth. Many prefer grey powder. The following is the routine treatment by grey powder adopted in the Army. Each pill contains one grain of grey powder. For one month the patients take six pills a day. Then they have three days' interval, and then for one month they take four pills a day. Then there are seven days' interval. For one month after that they take three pills a day, and then one month's interval. For three months three a day, then a month's interval; for three months two a day, then a month's interval; one a day for three months, then three months' interval; and then finally one a day for three months. In France the green iodide of mercury, half a grain doses, is used; that is rather more irritating than the grey powder. But I am certain that occasionally cases which do not yield to other forms of mercurial treatment quickly improve if you give green iodide. Lastly mercury as it is commonly given in the liquid form, as the perchloride of mercury. You know the liquor contains $\frac{1}{16}$ gr. in every drachm, so that one or one and a half drachms three times a day is a usual dose.

You will, of course, want to know why you should choose one of these methods more than another, for I may say that the old method of fumigation is given up now and therefore I have not mentioned it. The best is the intra-muscular. You know under that method exactly what you are doing, for you are certain that the drug is absorbed, and you know the patient takes it. But there are many cases in which that cannot be carried out, and in the case of a man like our patient, who leads a roving life, it is impossible, and the same is true of inunction, which, I think, is the next best. So with this patient, as with many others, we have to fall back on the treatment by the mouth. When he leaves London all we can do for him is to give him a prescription containing perchloride of mercury and iodide of potassium, and tell him he has to have it made up and take the mixture regularly for a couple of years.

June 13th, 1910.

TREATMENT IN SEPTIC CONDITIONS.

A Post-Graduate Lecture delivered at the Polyclinic, Chenies Street, on January 25th.

By EDRED M. CORNER, B.Sc., M.B., M.C., F.R.C.S.

THIS evening I should like to discuss with you a few points in connection with the treatment of the common septic conditions such as you must frequently have to treat. The reason why I personally have chosen this subject is because, during the last three years or so, I have had under my inspection about thirty-five of the beds at St. Thomas's Hospital which are devoted to patients suffering from surgical septic diseases. In that space of time many patients and a large amount of material has passed under my observation, and I thought the time ripe to gather together some observations upon these cases.

Curiously enough, the first point that I would make to-day is one which I met last; indeed, I did not realise its importance in the superlative degree until a few weeks ago. This point is the necessity of *rest*. We are apt to assume that rest is always made the prime condition of all treatment of acute conditions; but recently I had the point forced upon my notice that rest was not always enjoined as it should be. One of these cases, for instance, was that of a young woman who suffered from an ordinary septic leg. It was the kind of condition that would readily yield to rest and hot dressings. But she had been treated only with hot dressings, and had very little rest. After about a week or ten days it became evident that she was not getting on at all well, and was admitted to hospital. Here the hot dressings were continued, in addition to rest in bed, and she got quite well. The first point, therefore, is the necessity for rest in these acute cases.

The next point is that of *hot dressings*. There is no doubt that hot dressings, which are the modern and cleanly descendant of the old-fashioned poultice, do a great amount of good, chiefly by stimulating the circulation, not only in the part where the hot dressing is applied, but in other parts as well, and not only the circulation of the blood, but also that of the lymph. Probably they

also stimulate the activity of the cells of the tissues in the region affected. Therefore, the backbone of the treatment of septic diseases is, first, rest, and secondly, hot dressings. A large proportion of the cases get quite well under this simple treatment.

We can now proceed to the consideration of some further points which I have noticed in dealing with the cases brought to these septic beds. The percentage of the hospital cases in which an *incision* is necessary, in order to assist the hot dressings and the rest, is necessarily very high, and for this reason: that if the cases get well with ordinary care such as rest and hot dressings, the patients are as a rule not admitted to the hospital. These incisions have a number of uses. The most important use is allowing the escape of material, which, when retained, "clogs the processes of repair." That material—such as pus—removed, the part is better able to fight its own battle. But from time to time, indeed, very frequently when the patients are admitted to the hospital, it is necessary to do something more than these simple measures, and it is to these additions that I would particularly commend your attention.

One of these I have already mentioned in a lecture in this room, namely, the method of treating septic wounds and septic foci by means of *passive congestion.** The uses of passive congestion, like most other things, when it was first introduced was grossly exaggerated. It was even suggested that its use would exclude that of the surgical knife, which is, of course, quite untrue. Now that passive congestion has been in practice for some time, and its results can be submitted to calm criticism, it is found that the method of passive congestion has a number of failures. One of its failures is in the treatment of erysipelas, in which condition it does no good at all. The inflammation will travel as high as, or will even pass, the bandage which has been used to produce the passive congestion. And again, in a certain number of cases of septic infection of the joints and in gonorrhœal arthritis, passive congestion increases the amount of pain and appears to do more harm than good. Nevertheless, passive congestion is a form of treatment very easily applied, and possesses great advantages.

Let us consider it first as employed on the limbs. It is produced by applying a bandage around the upper part of the arm or the leg. That elastic bandage must not be tight enough in any way to interfere with the arterial pulse below it, and must only be sufficient to impede—not to stop—the return of the blood in the veins. It also impedes the stream in the lymphatics. As a result, the limb below the bandage becomes somewhat swollen. The difficulty in applying the treatment is the impossibility of finding any trustworthy guide as to how tight this bandage should be applied in a particular case. Indeed, the degree of tightness varies with every case, and almost with every application. In some people it has to be applied more tightly than in others. It may require two or three, or even half a dozen, applications of the bandage before the proper degree of tightness is obtained, and it is often found that the patient's statements are more trustworthy guides than the random attempts of the practitioner.

From the mention of this difficulty I pass to the consideration of the advantages which this form of treatment possesses. The great advantage lies in the fact that as soon as the right tightness is obtained, the medical man will know within an hour whether or not his treatment is going to do good. Sometimes the patient will lose his bodily pain within ten minutes of the application of the bandage. Previously the patient has been worn out with pain and sleeplessness, and, as a consequence, has no appetite. If that pain can be relieved—and it may often be relieved within ten minutes by this method—the patient will immediately drop to sleep, not the heavy unrestful sleep such as one sees in feverish patients with high temperatures, but a fresh, wholesome sleep, and on waking in, perhaps, two hours' time, he will feel much better, and, perhaps, take a good meal.

A further advantage is brought about by the impeding of the venous and lymphatic return by means of the bandage, while the arterial pulse is not affected. In the venous return the poisonous material is carried from the site of the disease to the body, and therefore, with the application of the bandage, the patient gets a far smaller dose of the poison than before. Consequently, within one or two hours the temperature will fall from, perhaps, 103° to 99° F. In a similar manner the pulse-rate will also become slower.

* Clinical Journal, vol. xxx, No. 6, 1907, pp. 94-96.

The treatment is easy of application; indeed, the patients can frequently carry it out for themselves, and altogether it is well worth trying.

Since people found that in certain cases—not in all—great benefit was derived from passive congestion as applied to the limb, it has been the ambition of many to apply the treatment by passive congestion to septic foci on the trunk, or neck, or head. It is quite obvious that bands cannot be applied to the trunk. Passive congestion has, however, been produced in the scalp by means of a bandage round the head, and in the face by a bandage round the neck. These are not methods which recommend themselves to patients, and moreover, from the clinical point of view, I have never seen such good results follow the application of the bandage round the head or neck as round the arm or the thigh.

Passive congestion is applied to the trunk by means of cups, really a revivification of the old method of "cupping" with modern appliances. A cup is used somewhat like an inverted wineglass with a hollow stem. Attached to that hollow stem is a rubber bulb, which, when squeezed, will expel the air, and the patient's skin having been anointed with vaseline or something similar, a vacuum is created inside the cup. By this means we can apply suction and a certain amount of passive congestion to areas of the trunk, but it is obvious that we can only do it for small foci.* But the cure of some septic foci on the trunk can be very greatly expedited by this method. Not only does the passive congestion itself help to relieve the trouble, but the suction also gives assistance to the patient.

Up to the present we have talked of passive congestion as combined with rest and hot dressings. I can now pass on to speak of passive congestion as combined with rest, hot dressings, and an incision. It is absolutely proved that with passive congestion, if an incision is required at all, it must be a very small incision. Whereas an incision of two or three inches would have been made in former days, now, with passive congestion, the patient may get quite well with an incision of half an inch. If, for example, the incision is made in the leg, and passive congestion is applied in the upper part of the thigh, certain changes will take place of which it is as well to be aware beforehand. The granulations or the tissues in the incision will become blue, cyanosed, and œdematous. I take special care to mention that result, because when people for the first time see the wound looking blue and pouting they receive an impression that it is worse than it is. To sum up, in medical cases which get well without incision, and in surgical cases in which incision is necessary, treatment by passive congestion can be employed, either by means of bands on the limbs or by cups on the trunk. And when an incision is made on the trunk there is the added advantage of suction.

I pass now to speak about the results I have found from modern methods of treatment, as, for instance, *vaccines*. Vaccines in the treatment of septic conditions have been employed for some years. I have seen them used in a large number of cases, so that I am in a position to form some estimate of their value. I may sum up the situation by saying that I am disappointed in the results. One of the things I hoped for when I undertook the inspection of these surgical infective beds was the possibility of formulating some conclusions as to the cases in which vaccines did good and those in which they did none. That hope has been unrealised. We have given vaccines in a large number of septic cases, some patients have recovered and others have not; and it is quite impossible to say whether the recovery in the one case and the non-recovery in the other are to be attributed to the vaccines. Doubtless some partisans of vaccine-therapy would say that in the cases which recover the result is due to the vaccines, and in the cases which do not recover the blame is to be laid upon the virulence of the infection. This attitude, however, I regard as not "sportsmanlike." Erysipelas is a septic disease, and vaccine treatment has been tried, amongst various methods, but it has not done the least good in the world. Similarly, the treatment of erysipelas by serums has been disappointing. We tried vaccines also with carbuncles. Carbuncles are usually due to a *staphylococcus*, sometimes to a mixed infection of a *staphylococcus* and a *streptococcus*. The vaccine treatment has not affected the small mortality from carbuncles. It may be asked whether it has lessened the duration of the disease and permitted an earlier recovery. If we desire to estimate the rapidity of recovery we may take as a

* For the breast see 'Operations' of General Practice' (Oxford Medical Publications), third edition, pp. 324, 325.

rough index the length of time which the patient has spent in the hospital. In St. Thomas's Hospital we have cases recorded for the last twenty or thirty years, both before the vaccine treatment and with the vaccine treatment; there is no appreciable difference in the time spent in the hospital by those who had vaccines and those who had not. There has not, therefore, been any particular success with vaccines in the treatment of carbuncles. With regard to the treatment of other septic conditions, septicæmia, pyæmia and septic bone diseases, the results have been equally indifferent. At first we gave vaccines in all such cases, without any obvious improvement, and afterwards we gave vaccines in only 50 per cent. of the cases in order to contrast them with those who did not have vaccines. The people who did not have vaccines got well just as quickly as those who had, so that on the whole the vaccine treatment has been disappointing. Of course in many cases vaccines were given and at the same time that an abscess was opened, when the temperature fell from, say, 103° to 99° F., who was to say whether the result was due to the vaccine or to the opening of the abscess? So far as one can ascertain, vaccine treatment may be of assistance to these patients, but it is certainly not a cure in itself. I remember a young doctor who had what turned out to be a *Staphylococcus aureus* infection of the hair-follicles at the back of the neck. He was perfectly willing to be a subject of experiment, and we put in a fine trocar and got out sufficient of the pus to make a culture. A vaccine was made from the growth, and he was vaccinated, continuing the experiment for a sufficient length of time to show conclusively that with vaccine alone we were not going to cure him. Surgical treatment was then resorted to, the boil incised, and the patient got quite well. That is not the only case in which the matter has been put to the proof in an experimental way, and it appears certain that if vaccine is to be tried it must be combined with surgical measures, in which case it is impossible to say clinically how much of the good result is due to the vaccine and how much to the surgical treatment. The reply that the upholders of vaccine treatment may make to this state of affairs is that the clinical results vary, of course, with the character of the infection. We make a practice of having a culture made from the patient's own vaccine, and in that connection we ran across one or two extraordinary things which helped to throw some distrust on the efficacy of this form of treatment. For instance, one can take the ordinary acne infection of the hair-follicles of the skin; some patients who have bad infections of the skin are admitted to the surgical infectious wards of the hospital, and have been vaccinated with a vaccine made from their own organism without any improvement. Afterwards one may give them a stock vaccine made from someone else's organism and they get quite well. As another instance, we may take gonorrhœal cases. If the patients are inoculated in turn from their own vaccine and from stock vaccine, in some cases their own vaccine will do good and in other cases the stock vaccine. In the majority of cases neither vaccine does apparent good. Therefore, it becomes a moot point as to how the character of the infection affects the vaccine treatment and its efficacy. But this much I will say, that I have never seen any harm follow the vaccine treatment, except when it has been continued for a long time, in which cases the patients have suffered from headache, anæmia, lassitude, and so forth. I can dismiss this part of the subject with a final statement. Having never seen harm follow the vaccine treatment, I would personally almost always use it, because after all it may do good, and it certainly does sometimes; and on that entirely negative ground its use may be advocated.

I turn now to the question of *septicæmia*, when the organisms are found in the blood. As a general rule we say that such a case will end fatally, but in recent times there has been an attempt to assist that septic patient by injecting a certain amount of fluid—say saline—into the loin or thigh. We know that if this fluid is injected into a certain part we create a focus of irritation there, and in consequence an abscess is formed from which we can grow the septicæmic organism. The enthusiasts who initiated this treatment called these abscesses *fixation abscesses*. They said that the organism was loose in the blood, and by means of "fixation abscesses" one could "fix" the organism, and so enable the patient to recover. We have tried this method, and have certainly obtained the septicæmic organism, and have seen the patient recover. But whether the determining factor in the recovery has been the abscess or the patient's own constitution it is difficult to say.

Pyæmic cases are certainly some of the most interesting that one meets in clinical practice. I have only time to give one or two instances. One is that of a big, heavy man, of forty-five to fifty, who was admitted to the hospital with a big car-

buncle in his neck.* He developed signs of pyæmia, the first sign being a swelling, first of one shoulder-joint and then of the other. When the first shoulder-joint was attacked we used the trocar and the aspirator, and from the fluid withdrawn grew an organism—the *Staphylococcus albus*. The joint was incised and drained. The right knee also swelled, and we were disinclined to make an incision. We tapped this joint and once more identified the pyæmic organism in the fluid. Rather to our surprise the man started to improve, and the knee-joint which we tapped, and from which we grew the organism, never refilled. During the convalescence the fluid appeared in the other shoulder-joint, which was tapped, the pyæmic organism again found, and the joint never refilled. The man entirely recovered. I saw him a year or eighteen months after his recovery, and found that the shoulder which we had tapped, and from which we had grown the organism, was a very good joint, and the knee-joint which had been affected was as good as the other knee-joint which had shown no signs of trouble. We have absolutely proved that although the organisms are present in these pyæmic foci they may get quite well. Not only may the patient recover, but the part also may recover its function. It has been known for many years that in pyæmia we get from time to time a localised swelling or a localised blush, and if it is left alone it disappears. Every now and then we have had these occurrences, and if the house-surgeon was available the swelling was incised and the culture taken, the pyæmic organism being grown from the fluid exuded. But if the house-surgeon was not available no incisions were made without apparently affecting the case. In pyæmia the body is full of organisms just as a sponge may be full of water, and these organisms may appear in a part and then disappear and the part get quite well. Without doubt a change will come over the treatment of pyæmic cases, and we shall incise the big foci, but confine ourselves to tapping and aspirating the small foci. There will be a number of men, particularly among those who have been able to watch a large number of septic cases, who will say that this is a retrograde step in the advance of surgery. But I do not think it is. We now know that what may be called medical rheumatism is a disease produced by a *streptococcus*, and rheumatic joints can get quite well without surgical treatment. Pyæmic joints are similar in character. In both instances the infection is through the blood-stream, and with pyæmic organisms in a joint that joint need not necessarily become disorganised. I argue, therefore, that in the future there will be a great change in the treatment of pyæmia.

My final point really opens up a big subject, but a subject which has been frequently thrust upon my notice when receiving reports upon these "septic" beds. We get *glycosuria* in septic cases, and it is quite a common thing in a patient who is suffering from a septic illness, particularly if he is run down in his general health, to discover this condition. When that glycosuria is suddenly discovered we are confronted with the problem as to whether it is diabetic or not. In the first place, it has been proved up to the hilt that almost every disease, particularly a septic disease, and I might even say every depreciation of health, is associated with the lowering of the "saturation point" of the tissues to sugar. We can take a healthy person, feed him with glucose, up to perhaps 1500 grm., and get a dietetic glycosuria. But with a person of lower vitality, only a dose of 1000 grm. might be necessary to produce the same result, and a person who was ill and whose resistance was less might show it at 100. Septic disease itself may cause the glycosuria from a patient's own diet or own tissues. There is a symptomatic glycosuria associated with septic diseases which may completely clear up as the patient improves. It happens from time to time that a patient is brought into the hospital, the urine tested, no sugar is found, and he is put on the ordinary hospital diet. In a week or ten days the house-surgeon may report that the patient has a large amount of glycosuria. For instance, in a case at present in the wards the amount of sugar is now so great that not even a careless house-officer could have missed it, and yet in this case it was reported when the patient was admitted that there was no glycosuria. The fact is that in the hospital the "septic" patient may get a dietetic glycosuria, which will clear up before the patient leaves the wards. The occurrence of the glycosuria is a distinct indication that the man's general condition is depreciated, and probably that the septic disease from which he is suffering is worse. The indication, therefore, is for active treatment. In other cases the glycosuria is due to a definite disease called diabetes, and in these cases I would also urge that its occurrence is again an indication for active treatment, and the earlier and the more energetic the treatment undertaken in diabetic cases the better for the patient. It is not worth the doctor's or the patient's while to expend a large amount of time and pain to save a foot, for example, which is affected. It is a great deal better from the point of view of the patient's general health that an amputation should be done. In such a case the sugar in the urine, which was from the diabetes, becomes markedly less in amount, and the patient's general health is very greatly improved. The result is far better than if the patient underwent a long illness in the hope of staving off amputation. As a general rule, therefore, the discovery of sugar in the urine is an indication for more energetic treatment.

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TWO CLINICAL LECTURES ON HEMIPLEGIA AND ITS TREATMENT.

Delivered at the National Hospital for the Paralysed and Epileptic.

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LECTURE II.

GENTLEMEN,—You will remember that in our last lecture we dealt with the more or less typical features of hemiplegia, and we dealt with hemiplegia as such, that is to say, the condition in the narrowest restriction of the term, namely, the loss of power on one side of the body due to a lesion of the cerebro-spinal tracts which take origin in the opposite hemisphere of the brain.

To-day I shall show you a few cases to illustrate other aspects of hemiplegia, and deal with the symptoms which a lesion in any portion of the cerebro-spinal tracts may produce. You will easily understand that the lesion that interrupts the pyramidal tracts may also involve the optic thalamus or many of the intrinsic centres of the mid-brain, pons Varolii, medulla oblongata, or even in the upper segments of the spinal cord, and thus produce additional symptoms, or modify those of ordinary hemiplegia. I have not cases to illustrate all these conditions to you, but those I can show you I think will be instructive.

The first case is that of a woman, æt. 48 years, who was well until February, 1904, when she had an attack of complete paralysis of the left limbs, which set in during sleep. Power began to return in the leg fairly rapidly, and she was able to walk about in from one month to six weeks, but the return of power in the arm was very much slower. In the history which she gave us she said that for a time, while she was unable to move the left fingers or the left arm, these fingers moved spontaneously, and as she sits here you will see certain involuntary movements of the limb of a curious type. When she came to see me here in the Out-patient Department last August, the symptom of which she complained chiefly was not the loss of power or inability to use the arm, but the intense pain which she suffers down the whole of the left side of the body. I remember that although it was a warm day and the windows were consequently open, when she was being undressed she asked that the window might be closed, as a draught of cold air on the hand caused a pain to start, which radiated over the whole of the left side of the body. Though this pain has been her chief symptom, she complains of other disagreeable sensations in the same side of the body and limbs. The pain extends over the whole of the left side of the body, trunk, face, and limbs; it is never completely absent, but becomes much aggravated by various forms of external stimuli affecting this side of the body. Under usual conditions it is "an aching, like severe neuralgia," but frequently it becomes much more severe, returning after a time to its usual intensity. At one time this patient describes this pain as if someone was cutting her and rubbing salt into the wounds, at another, as if she had been crushed between two boards and was very sore after it, or she speaks of it as being an "intense burning sensation." The pain is usually worse on the outer sides of the limbs, and in the face on the left side of the nose. It is also present on the left corner of the lips, but not in the mouth, or tongue, or in the eye. But it frequently radiates widely over the left side. Then there are certain paths along which it seems more liable to travel; they are illustrated in this diagram. For instance, by applying a cold test-tube to the side of the face we may start pain, which radiates down to the neck, to the point of the shoulder, and thence spreads down the back, but it may start in any part and radiate in any direction. The chief external stimulus which excites the pain is cold. She dare not attempt to wash her hand in cold water, and handling cold objects with the left hand causes her much discomfort. This pain is not present on the normal side.

On further examining her we find all the ocular movements are intact, but there is very slight nystagmus on lateral movement of the eyes. Both sides of the face move equally on both voluntary effort and emotional display. Her tongue is also protruded straight and the palate moves symmetrically. But there is some paresis of the left arm, and adhesions in the shoulder-joint limit its movements very much, but if she persists in any movement she can execute it always to full range. The

left leg is also weak, and she walks in a hemiplegic manner, holding the limb stiffly and dragging the foot a little. But one of the typical symptoms of ordinary hemiplegia is completely absent, for there is no rigidity or tendency to contracture; I can bring the hand and forearm and fingers into the same plane, and extend the fingers on the forearm further than on the normal side. It is the same in the other segments of the limbs. The other symptom, which at times is very marked, is spontaneous and involuntary movements which occur in this hand; they belong to the type which is known as athetosis, the predominant movement of the fingers being extension at the metacarpo-phalangeal and the first phalangeal joints. Sometimes these movements are absent for a time, or more irregular movements of a choreic type may replace them. Her vision is as good as ever it was, there is no hemianopia or hemiachromatopia. She is slightly deaf in the left ear, especially for the highest notes, and the tone-range is not as good as in the right ear. This slight deafness probably developed with the hemiplegia. Merle has recently observed it in other cases of thalamic lesion.

But the most striking symptoms we find in this case are when we test the sensibility. On examining tactility with cotton-wool we find she feels contacts as well on one hand as on the other, and even on the hairless parts there is no difference in sensibility to touch. Secondly, she can localise perfectly the touches she appreciates, but when we test her power of discrimination between one point and two of the compasses we find there is a slight defect in the left hand. For instance, on the normal palm she can discriminate the two points at a distance of 1·5 centimetres, but it is necessary to separate them to 3 centimetres before she can recognise both on the left palm. Much more prominent is the phenomenon which we get on testing sensibility to pain and temperature, as a pin-prick is much more painful to her on the affected side, and the pain it produces does not remain localised, but spreads a considerable distance around the point that is pricked. Further, the pain which the stimulus evokes may persist for several seconds. The same holds true for other forms of painful stimuli. On the other hand, when we measure the threshold of the pain, we find that it is raised on the affected side, as a stronger stimulus is necessary to produce pain on this side; with a pressure of 3 grammes on the needle of this algesimeter its point feels dull on the affected side, but it pricks on the right side. This is a very characteristic feature of thalamic lesions, to find the threshold to pain somewhat raised, but that when a painful stimulus is effective it is more painful to the patient than the same stimulus is on the normal side. We find much the same condition on testing sensibility to temperature, for you would see if I were to take you through a long series of tests on the left side of the body, that she would not appreciate the mean degrees of temperature as well as on the right side, while the higher degrees are readily recognised and not confused. But all temperatures below 20° C. and above 50° C. are extremely unpleasant on the left, much more so than on the normal side, and they frequently excite pain that may radiate far from the spot touched, or even extend over the whole of the left side. For instance, touching her left hand with this test-tube that contains water at about 50°C. excites a pain that extends to her elbow, while the tube is not uncomfortable to her right hand. Further, I can show you that there is considerable loss of the sense of position in the left arm, and the sense of passive movement in that limb is very imperfect. Though touch, as we have seen, is unaffected, tickling produces an extraordinarily uncomfortable sensation on the left side of the body; for instance, the patient objects very much and gives unmistakable evidence of discomfort if I tickle the sole of her foot.

This case of hemiplegia is distinguished from the two cases I have already shown you by the much greater affection of sensibility, both subjective and objective, in relation to the degree of paralysis that exists. It represents to you, in almost its most typical form, the "thalamic syndrome." We have been familiar for years with the cases in which the chief symptom is pain in the paralysed side of the body, or various other uncomfortable sensations of hemiplegic distribution, but we knew very little about the localisation of the lesion that produces them and the course of the symptoms until recently, when Dejerine and Roussy drew closer attention to them. Roussy has pointed out that there are certain symptoms which characterise this syndrome that results from lesions of the optic thalamus. In this rough diagram you see the motor fibres of the pyramidal tract pass through the internal capsule beween the optic thalamus on

the one side and the lenticular nucleus on the other. Consequently a lesion in the optic thalamus is very likely, if it be large, to extend to, or involve, the internal capsule as well; so we find in most of those cases of thalamic lesions that there is in addition a definite hemiplegia, but it is frequently slight. The thalamic syndrome is characterised by the fact that contractures are rarely present with even severe hemiplegia, and, as in this woman, the Babinski sign is frequently absent despite the presence of some hemiplegia. It is difficult to test this woman's plantar reflex owing to the intense discomfort produced by tickling the sole of her foot, but I have never obtained an extensor response. The sensory symptoms are more characteristic. Roussy has laid stress on the constancy of *hemianæsthesia*, which, according to him, involved deep sensibility more than the cutaneous forms in most of his cases, that is, there was a relatively greater affection of the appreciation of position and of movement than of tactile, pain or thermal sensibility, but, as you have seen in this woman—and the same is true for other cases I have observed—there is but little loss of cutaneous sensibility, and the sensibity of the deeper structures of the limbs is not more severely affected. A second feature of thalamic lesions is the presence of severe *paroxysmal pains*, which are usually spontaneous, but may be excited by sensory stimuli; this is the most characteristic and perhaps the most serious of the symptoms. The agony which this woman suffers is scarcely measurable, and other cases have committed suicide owing to it. A complaint of pain in the paralysed limbs is not uncommon in hemiplegia, but we must distinguish two different varieties; the commoner form is a dull aching, which is generally referred to the neighbourhood of the joints and is probably associated with changes in them; it is increased by movement of the joints. The other form is that which we find in cases of thalamic lesion, as in this woman. It is often almost constant, but varied by severer paroxysms. The worst feature is that little can be done for it so far as we know at present. It is of central origin, and no drug that we know has any influence on it; cutaneous applications are worse than useless, as any form of irritation of the affected limbs only increases it. Opium has relieved the pain in some of the cases I have had. The third symptom on which Roussy insists is *hemiataxia*. We have already seen that there is more inco-ordination of voluntary movement in my patient than in ordinary hemiplegia. A fourth feature is the presence of *choreiform or athetoid movements* in the paralysed limbs; in some cases the movements are irregular or choreiform and in others they are more athetoid, or, as in the next case I will show you, the spontaneous movements take the form of tremor. Occasionally this thalamic syndrome is associated with hemianopia, but this is rare, as the lesion that produces it does not extend into the pulvinar or into the retro-lenticular segment of the internal capsule. In many text-books you will find reference to Nothnagel's sign, or the greater weakness of one side of the face in emotional than in voluntary movement; apparently it results from a thalamic lesion, but it was not present in Roussy's cases, or in marked degree in any of those that I have seen; probably the lesion that produces it does not occupy the same site as that which produces the thalamic syndrome.

I show you now the second case, through the kind permission of Dr. Collier; he is a man, æt. 43 years, though his appearance suggests that he is much older. He has led a hard life, but was in good health until early in January this year. Then he had an attack of weakness on the right side, which set in with a feeling of numbness and tingling and an uncomfortable and unnatural sensation on the whole of the right side of the body. There was no loss of consciousness with the onset of the attack, and there was never much paralysis, in fact, he was able to walk home immediately after the stroke. He has not had much severe pain, but sometimes complains of aching on the affected side of the body; this presents all the characters of the pain in the last case which I showed you. His special senses are normal, he has no hemianopia, and he hears equally well with the two ears. There is no defect in any of the ocular movements, but when the attack first came on he had diplopia. You see, however, the right pupil is very much larger than the left, in fact, almost twice its diameter, and it fails to react to light and contracts only very slightly on accommodation; the reaction of the left pupil to both light and accommodation is sluggish. When he shows his teeth and smiles the movements are equal on the two sides of his face, but you may notice a constant tremor at the right angle of his mouth.

His speech has never been affected. The power of movement in his right arm is surprisingly good considering the attitude in which the hand is held, and the range of movement is unrestricted. An ordinary hemiplegic of that type which I showed you on the last occasion would never be able to extend the fingers as this patient does. You notice, too, that we can over-extend the fingers and the wrist, so that here, instead of having rigidity, there is a diminution of tone in the affected limbs, which offer practically no resistance to passive movements. There is only slight weakness of his right leg. You will notice at once that there occur spontaneous movements of the right arm, both of the limb as a whole and of its different segments. These movements vary a good deal in character; when he allows his hand to hang loosely these generally take the form of a more or less rhythmical tremor of the wrist or fingers, but sometimes they are more irregular and can be described only by that undesirable word "choreiform." There is also considerable inco-ordination of the voluntary movements of the affected limbs and marked intention tremor; you can see it when he attempts to place his right forefinger on his nose, and it is even more marked when he attempts to touch an external object. All his reflexes on the affected side are brisk, and plantar stimulation gives an extensor response. He complains chiefly of numbness, tingling and pain down this side of the body, and of an unnatural feeling which is very uncomfortable to him, but he is unable to describe to us what it is. On testing sensibility we find that he fails to appreciate many touches with cotton-wool on the right hand, and, in fact, on the greater part of the right side of the body, while he reacts to every touch on the left hand; he also fails to localise correctly contacts that he can feel on his right side. When I attempt to tickle him by drawing my fingers or any suitable object over the skin on the two sides of his body you will observe a remarkable difference in his reaction from the right and the left side, for while on the left side the stimulation is not disagreeable to him, on the affected side it produces intense discomfort, of which he complains bitterly; owing to this it was scarcely possible to test his plantar reflexes, and he tells you that on the hand it starts an uncomfortable sensation that runs up the arm. As in the last case I showed you, it requires a stronger stimulus, whether pin-prick or deep pressure, to produce pain on the affected than on the normal side, but when pain is produced it is much greater, and spreads from the spot at which it is produced. As in that case, too, extremes of temperature produce pain or extremely unpleasant sensations. Finally, there is a marked defect in the appreciation of the position in which the limbs lie.

Here we find certain features different to what we found in the first case. In the first place the pupils are unequal, and their reaction to light and accommodation is absent or much reduced. We know that the third nerve nucleus lies immediately underneath the optic thalamus, and that the most anterior portion of this nucleus contains the centre that subserves the pupillary reactions; consequently any damage to it may explain this symptom. A second feature in this case is the greater loss of the different forms of sensibility; he fails to recognise cotton-wool touches on the right hand. The next is the presence of tremor and irregular movements on the affected side of the body. What is the significance of these tremors and irregular spontaneous movements? Such a tremor as you have seen in this man is not uncommonly found in lesions of the upper portion of the mid-brain. In many cases it is very like that which is found in paralysis agitans, but often is more irregular. There has been much written on this subject; some years ago I attempted to connect it with lesions involving the red nucleus, which lies immediately under the optic thalamus, or the connections of this nucleus. You will remember that from each lateral lobe of the cerebellum there come fibres through the superior cerebellar peduncle to end in the red nucleus of the opposite side, while fibres that take origin from this nucleus at once decussate across the middle line, and pass down to that side of the spinal cord; thus we have this double path, the cerebello-rubro-spinal path, and there is evidence that a lesion in any part of that may produce two symptoms—rigidity of paralysis agitans type, and tremor. Therefore, the presence of this tremor and the defect in the pupillary reactions suggests that in this case the lesion extends to the lower portion of the optic thalamus or the upper part of the mid-brain, and we consequently have the sensory symptoms of a thalamic lesion associated with those of a lesion in the upper part of the brain-stem.

The symptoms that result from lesions in other parts of the brain-stem are characterised chiefly by the presence of certain cranial nerve palsies. If, in this man whom I have just shown you, the lesion had been slightly lower, it would have produced paralysis of the oculo-motor nerve of the left side with right-sided hemiplegia: that is the condition of superior alternating hemiplegia, or Weber's syndrome. Or if a lesion involve the middle of the pons it may interrupt the pyramidal tract, which innervates opposite limbs, and at the same time injure the nuclei or the roots of the fifth nerve and produce paralysis of the masseter and pterygoid muscles on the same side, and anæsthesia of that side of the face. While if the lesion lies lower in the lower pons you would have the most common of these crossed hemiplegias, with the seventh nerve involved and consequently peripheral facial paralysis on the side of the lesion and paralysis of the opposite limbs. Similarly, lesions in the medulla may produce characteristic cranial nerve palsies with weakness or paralysis of the opposite limbs. Time will not permit me to attempt to illustrate these various symptoms to you, but I would finally like to show you a case which is of considerable interest.

He is a healthy man, æt. 32 years; he was a soldier in the South African War, and returned in good health. But in May, 1908, a motor bus ran down a van he was driving and threw him to the street. He became unconscious immediately, and lay so for the next three weeks. When he regained consciousness he was lying in the hospital, and was unable to move the right arm or right leg, but he was not at that time aware that there was anything else wrong with him. He gradually improved, and lately came to Dr. Collier's out-patients', and he has kindly allowed me to show him to you. You can see at once all the symptoms of a right hemiplegia, but you must notice that the right side of the face is not involved, and none of the other functions of the cranial nerves are disturbed, and he never had any affection of speech. All the reflexes of the right side are such as you get in ordinary hemiplegia; the arm-jerks are extremely brisk on the affected side compared with the other, the knee-jerk is much brisker on the right side than on the left, and one can easily elicit ankle-clonus on that side. There is also an extensor response to plantar stimulation on the sole of the right foot, while on the left side the normal flexor response is obtained. But on examining him more closely we discover that there is much more than the right hemiplegia, and I shall ask your attention to what may be found on examination. You will see at once that even the slightest touches are recognised on both sides of the body, and further, that he can localise all the touches, which he feels perfectly well on both sides.

But on testing sensibility to pain with a pin-prick we find that though it is normal on the paralysed side it is completely absent on the left limbs and the left side of the trunk; on both sides of the face, however, painful stimuli are equally acute. The appreciation of pain produced by all other forms of stimulation is equally lost. As I measure deep sensibility with Cattel's algometer you may see that a pressure of five kilogrammes on his right palm produces pain, while I fail to give him any discomfort with a pressure of even twenty kilogrammes on his left side. On testing thermal sensibility, too, we find that though all degrees of temperature are normally recognised on the right side, on the left side test-tubes containing even ice, or water at 55° C., are only recognised by the patient as tactile stimuli. On the other hand, I can easily demonstrate to you that the sense of position and of passive movement in the paretic limbs are very defective, in fact he has very little idea of the position these limbs occupy in space, while on the left side these forms of sensibility are unaffected. Finally we find that on the right side he is unable to recognise the vibrations of a slowly vibrating tuning-fork, or discriminate between one and two points of Weber's compasses when they are separated to a distance at which he can easily discriminate between them on the left side.

Thus we have on the right side spastic weakness, associated with the loss of the sense of position and of movement, and inability to appreciate a slowly vibrating tuning-fork and to discriminate between one and two points simultaneously touched at a short distance apart, while the power of the left limbs is intact, but the appreciation of pain and temperature is completely lost on this side of the body. This is the condition generally known as the Brown-Séquard syndrome, though in it we frequently find that tactile sensibility is affected on the same side as thermal and pain sensation. It can be produced only by a unilateral lesion of the spinal cord, and the fact that there is no weakness of the facial muscles or other muscles supplied by the cranial nerves, and that the analgesia and thermo-anæsthesia do not extend to the face, proves that in this man the lesion must lie in the highest segment of the spinal cord. The case shows you that from a spinal lesion we may have sensory and motor paralysis dissociated, hemiplegia on the one side with sensory loss on the opposite side. This condition cannot be produced by any lesion that lies above the level of the spinal cord.

June 13th, 1910.

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CONTENTS.

PAGE

***Foreign Bodies in the Air-passages.** By CECIL WALL, M.D., F.R.C.P. ... 161

***The Treatment of Indigestion.** By E. I. SPRIGGS, M.D., F.R.C.P. 173

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FOREIGN BODIES IN THE AIR-PASSAGES.

A Clinical Lecture delivered at the London Hospital.

By CECIL WALL, M.D., F.R.C.P.,

Assistant Physician to the Hospital.

WHEN a solid foreign body of appreciable size passes into the air-passages, the effect as a rule is sufficiently startling: if it is not expelled, the result, immediate or remote, is likely to be untoward.

Historical.—It is curious to note how gradual has been the evolution of the treatment of this accident: the earliest recorded example (1) is that of the poet Anacreon, who at the age of eighty-five was choked by a raisin some 600 years B.C.

Thenceforward, until the eighteenth century, as Louis (2) plaintively remarks, "the majority of the cases handed down to us by former practitioners of foreign bodies which have passed into the respiratory tube contain little more than the recital of the melancholy ending to which the accident had given rise."

It is true that in 1620 Habicot, of Paris, performed bronchotomy successfully on a boy, æt. 14 years, for the relief of dyspnœa caused by the impaction of a packet of gold coins in the pharynx, and Monavius, of Halle (who, according to Louis, translated Habicot's book, and without acknowledgment published it as his own in 1644), recommended bronchotomy as a means of removal of foreign bodies from the air-passages; but even in 1759 Louis complains that the approved method of treatment was by emetics and sternutatories. He relates a case of his own in which he was prevented by his colleagues from performing tracheotomy to save the life of a little girl, æt. 7 years, who had inhaled a haricot bean into the trachea.

In 1731 Juncker (3), in his conspectus of surgery, says that some people recommend bronchotomy for the removal of foreign bodies which have fallen

into the trachea; it is said that this is the first reference to the mode of treatment made in any systematic treatise on surgery. Juncker expresses no personal opinion for or against the treatment.

After Louis's paper the operation of tracheotomy (or bronchotomy, as he called it) was fairly frequently performed. The simple procedure of inversion of the patient does not seem to have been suggested until in 1843, when the famous engineer Brunel (4) accidentally inhaled half a sovereign into his trachea. The inversion was accomplished on a special table of Brunel's own invention, but was not successful until a preliminary tracheotomy had been performed. I cannot find an example of successful inversion of the patient without tracheotomy earlier than the case recorded by Sir Thomas Watson (5), who relates how Dr. G. B. Halford, when house-surgeon at the Westminister Hospital in 1852, stood a patient upon his head in the corner of the receiving room.

Tracheotomy, with or without inversion, was considered the best treatment until quite recently, though Weist (6), in 1881, after analysing 1000 cases, concluded that the operation should not be performed except in cases of great urgency. In 1894 Kirstein (7) introduced the method of direct laryngoscopy, and in 1897 Killian (7) was the first to pass a rigid tube into the left bronchus and thus successfully remove a foreign body.

Since Killian's brilliant work was published, bronchoscopy, either by the inferior or superior route, has been recognised as a valuable method of seeking and removing foreign bodies from the air-passages.

Cause of accident.—The accident of inhaling a foreign body usually occurs in a moment of surprise and during the act of inspiration, the patient being often enough in perfect health. Rokitansky (8) records a curious case of a small boy who was playing with a blow-pipe and inhaled a feathered dart with the point uppermost. The dart fell into the left bronchus and caused a paroxysmal cough. Twelve days later the boy died of hæmorrhage, and it was found post mortem that the point of the dart had been forced by coughing against the right wall of the trachea, and had penetrated through this into the innominate artery.

Contributory causes.—Sometimes there may be a contributory cause such as local anæsthesia or paralysis of the larynx and pharynx, or general anæsthesia of the patient caused by drugs or disease, or there may be a permanent tracheotomy opening.

Alcohol.—The cases in which there is anæsthesia are numerous: Alcoholic intoxication may be responsible. Jackson (9), in 1844, recorded a case in which a man, æt. 31 years, who had been put to bed drunk, was found dead shortly afterwards with a thin, transparent piece of potato-skin across the rima glottidis.

Nitrous oxide.—Stumps of teeth, adenoid growths, tonsils, etc., may fall into the trachea during the anæsthesia induced to facilitate their removal. I shall relate an example later.

Epilepsy, apoplexy, uræmia. — Foreign bodies may pass into the trachea when the patient is unconscious owing to disease, such as epilepsy, apoplexy, or uræmia.

Case 1.—In 1909 a man was admitted to the London Hospital under the care of Dr. Percy Kidd suffering from chronic interstitial nephritis and uræmia; it was noted that his teeth were carious and loose. At the autopsy a tooth was found in the right bronchus and the lung was in a condition of septic broncho-pneumonia.

Suicide.—The foreign body may have been introduced into the air-passages intentionally, and not accidentally.

In 1842 a woman died suddenly in prison. A brief post-mortem examination was performed, and a medical certificate of death from apoplexy was signed and the body handed over to the anatomists. In the dissecting room it was found that death had been caused by a plug of spindle-cotton (10) which blocked the throat firmly, and evidently had been introduced by the patient herself with suicidal intent.

Murder.—In 1855 Littlejohn (11) recorded a case of a woman who had died suddenly. On examination he found a wine-cork inserted firmly in the upper part of the larynx. Some ribs were fractured, and it was obvious that the death had not been natural. At the trial it was suggested that the woman, whilst extracting the cork from the bottle with her teeth, might, by the sudden impetus of the contained fluids, have drawn it into the position in which it was found.

Littlejohn pointed out that the sealed end of the cork roughened by the passage of the cork-screw was uppermost in the throat, and thus disposed of the theory of the defence. The cork

had been forced into the larynx while the woman was in a helpless state of intoxication.

Littlejohn also records examples of infanticide by dough (12), and by strips of newspaper (13) forced into the larynx.

Nature of foreign body. — A search through the literature soon demonstrates the enormous variety of foreign bodies which have been found in the air-passages; the only common factor is that the body is small enough to pass into the larynx. Bodies with a diameter of 1 in. may sometimes pass into the air-passages of a man; in the female $\frac{5}{8}$ in. seems to be the usual maximum diameter, and in young children it may be as small as $\frac{1}{5}$ in.

From the point of view of prognosis and treatment Killian's classification is of some value.

The bodies may be grouped into those that are hard and those that are not hard.

The hard bodies may be *smooth and spherical*, such as peas or marbles, cherry- and plum-stones, bullets, etc.; or *smooth and not spherical*, such as coins, buttons, collar-studs, dress-hooks, tracheotomy tubes, etc., or they may have *sharp points which engage in and lacerate the mucous membrane* — for instance, needles, pins, nails, nibs, teeth, artificial dentures, nut-shells, pieces of bone, tracheotomy tubes, darts.

The bodies which are not hard may swell with moisture—for instance, pieces of maize leaf or other vegetable substance, beans, peas, etc.; or they may be *unaffected by moisture*, such as pieces of wood or cork, orange- or melon-pips, fruit seeds, pieces of down, cotton, meat, etc.

There is an interesting story of a pea which remained for some time in the trachea of a boy, and when rejected was found to be germinating, if not bursting into bloom. I have tracked this story through all the literature of the subject, but have been unable to verify the original narrator. It is told by Schurigius (14) in his 'Chylologia,' published in 1725, and he gives a reference to Riedlinus (15), who published his medical observations in 1691. The Royal Society of Medicine possesses the only copy of Riedlinus of which I can find evidence, and unfortunately, owing to the removal of the library, the book cannot be obtained at present. Schurigius says that the boy inhaled a pea; symptoms persisted for some years, and the doctor in charge abandoned treatment. His mother was recommended by a friend to try an infusion of black cherries as an infallible specific. She sent the apprentice to get some from the local druggist's, but the dispenser in error gave aqua fortis. The mother, all unsuspecting, administered a spoonful; the dire result necessitated another visit from the doctor, who administered olive oil as an antidote. The boy vomited, ejected a pea, "jamjam germinans," and rapidly recovered. Schurigius does not recommend a repetition of the treatment by sulphuric acid in similar circumstances.

There is one class of soft foreign body which is of peculiar interest: *an ear of wheat, barley, rye, or other grass* inhaled stalk end downwards tends to penetrate toward the lung with every movement.

Thus Sir Thomas Watson (16) demonstrated at one of his lectures an abscess common to the right lung and the liver, which contained an ear of rye. The patient, a coachman, was sucking the ear as he drove; the carriage gave a sudden jolt, and he inhaled it. There were no immediate symptoms, but some time afterwards hectic fever with fœtid expectoration developed and continued until death.

Mr. Godlee (17) has recorded two cases in which a head of grass worked its way through the chest-wall.

And Dr. Good, in the 'St. Bartholomew's Hospital Reports' for 1891, recorded a similar case in which a head of grass appeared three inches below the axilla in a child of five.

In estimating the value of Killian's classification, it must be remembered that the secondary effects of an inhaled foreign body may be due to the adherent micro-organisms rather than to the physical characters of the body. Thus it is conceivable that Sir Thomas Watson's case just quoted was complicated by infection with the actinomyces.

Symptoms.—The results of the inhalation of a foreign body into the air-passages are generally immediately manifest; if the body is not rejected, and the patient survives, further secondary or remote consequences may ensue.

The immediate consequences vary from complete absence of symptoms to sudden death.

Hechstetter (18) records the case of a citizen of Augsburg, who, under the notion of curing a pain in his teeth, put a Portugal ducat into his mouth. On waking he found the piece gone. As he

swallowed easily, however, and moved his tongue freely, he thought he must have swallowed the money; but some time after, finding himself becoming hoarse and getting thin, he consulted several physicians and surgeons, who could not reach the foreign body with their fingers or with any instrument. At the end of two years and two months he brought up the ducat again. Louis, who relates the case, assumes that the ducat must have remained during this period in one of the ventricles of the larynx.

Mackenzie (19) in 1851 recorded an example of a very different result. A man, choked while eating his dinner, walked across the road to a chemist's shop in the hope of obtaining relief and fell dead as he entered it. At the autopsy a piece of beef was found resting on the glottis, pushing forward the epiglottis, and by its lower extremity entering and occluding the rima glottidis.

Dr. Percy Kidd told me that he once performed an autopsy at the London Hospital on a man who had been seen to fall in the street at Aldgate as though in an apoplexy. No abnormality was discovered, except that a large piece of orange-pulp was found resting on the rima glottidis.

Peas, oysters, gooseberries, and even a piece of tough mucus have been known to cause sudden death in the same way.

I have already mentioned the case of Anacreon, who was choked by a raisin some 2500 years ago.

When sudden death has resulted it has usually been found that the foreign body has obstructed the glottis; if the substance passes the cords and reaches the trachea or bronchi an immediately fatal result is much less likely to occur.

Most commonly symptoms of severe respiratory obstruction follow the inhalation of a foreign body; these may persist if the substance remains in the undivided air-way, or abate if it drops into one or other bronchus; movement out of the bronchus may then produce paroxysmal attacks of dyspnœa. The resting-point of the foreign body, therefore, is a matter of importance.

Case 2.—In 1898 a small boy, æt. 2 years, was brought to the London Hospital asphyxiated, cyanosed, and without respiratory movement. Digital examination of the pharynx revealed a marble impacted in the rima glottidis. The marble was removed, artificial respiration performed, and rapid recovery followed.

Case 3.—In August, 1900, a boy, æt. 7 years, was admitted to the London Hospital, under the care of Mr. Hurry Fenwick, with the history of having swallowed a fish-bone. There was some stridor, and paroxysmal attacks of dyspnœa occurred. The bone could not be seen on laryngoscopic examination. Tracheotomy was performed, with relief to the symptoms. Five days later a fish-bone, one inch long, with a sharp point, which had been impacted in the larynx, was removed by the finger. Recovery then ensued.

Foreign bodies once past the larynx usually fall into a bronchus; occasionally jagged bodies stick in the trachea.

Case 4.—In 1903 a girl, æt. 2 years, was admitted to the London Hospital under Mr. Hutchinson, with markedly obstructed respiration and a history of having swallowed something. Digital examination under an anæsthetic failed to reveal anything and consequently a low tracheotomy was performed. A piece of nutshell was found impacted in the trachea just above the incision. This was removed and rapid recovery ensued.

Case 5.—At the end of this lecture I will show to you a small boy who was admitted to this hospital last year under my care, suffering from severe laryngeal obstruction: the parents stated that they thought he had swallowed a piece of bone the wrong way. Immediate tracheotomy relieved the dyspnœa, but no evidence of any foreign body could be obtained by the X-rays or otherwise, and the trouble was thought to be due to œdema of the larynx. The obstruction, however, continued, and it was impossible to remove the tracheotomy tube. At length after two months, Dr. Lack, with the aid of the bronchoscope, found and removed a sharp piece of bone which was impacted just below the larynx. Granulation tissue now narrowed the trachea, and it was not until seven months later that the tube was permanently removed.

Stokes (20), in his 'Diseases of the Chest,' published in 1837, records the case of a foreign body in the larynx in which the symptoms were somewhat peculiar. A boy was playing with a whistle which had been carved out of a plum-stone; this slipped into the larynx but caused no discomfort. For three days the boy ran about, pleased with his power to produce sounds from the invisible whistle: when he began to get attacks of suffocative cough he was taken to the hospital. Laryn-

gotomy was performed and the whistling ceased, but began again when the wound closed. Later the plum-stone fell into the right bronchus and was ultimately recovered through a tracheotomy wound.

If the foreign body does not lodge in the larynx or trachea it usually falls into the right bronchus, because the promontory between the bronchi is slightly to the left of the middle line; this rule, however, is not absolute. In Rokitansky's case, previously quoted, the puff-dart fell into the left bronchus; later I shall relate to you several other instances. Sometimes a foreign body falling into one bronchus may be displaced by the act of coughing and then pass into the other.

McPhedran, in Osler and Macrae's 'System of Medicine,' relates the case of a seven-year-old boy who inhaled, while at play, a small revolver bullet. At first there were some slight symptoms of respiratory distress, but after a few hours there was only slight dyspnœa, increasing somewhat on exertion. A few days later bronchitis, pneumonia, and right pleural effusion developed, and the boy became very ill. Ten days later the temperature became normal and the symptoms abated, though cough persisted. A few days later the pyrexia recurred and the cough became paroxysmal. From the onset the breath-sounds had been weak on the right side. Pain now developed in the left side, and the breath-sounds were weak over the left lower lobe. An examination with the X rays showed the bullet to be on the left side. Tracheotomy and inferior bronchoscopy were performed, and the bullet was extracted from a small branch of the left bronchus; rapid convalescence ensued. McPhedran has little doubt that this case is an example of a heavy substance like a bullet being forced by the act of coughing out of one bronchus and falling back into the other. It is obvious that a foreign body passing into a bronchus will lodge in the smallest branch which will accommodate it.

The symptoms and signs produced by a foreign body in a bronchus are determined by its size and physical characteristics. Killian's classification is again important.

Hard bodies, smooth and spherical, such as peas, marbles, fruit-stones, bullets, etc., tend to occlude the bronchus in which they are impacted. The degree of the resultant dyspnœa will depend on the amount of lung put out of action.

Cough results from mechanical irritation of the bronchial mucosa, the intense sensitivity of which is shown by the need for local cocainisation in the operation of bronchoscopy, even when the patient is in deep chloroform narcosis.

The signs at first are those of defective air entry into a part or whole of the lung and later of pulmonary collapse, or perchance of septic infection, which leads to bronchitis or bronchiectasis with retained secretion.

McPhedran's case which I have just quoted is a very good example of this group.

Hard bodies which are not spherical do not necessarily in the earlier stages lead to complete occlusion of the bronchial lumen: secondary swelling of the mucous membrane may cause absolute obstruction.

If they are smooth, such as coins, buttons, whistles, collar-studs, dress-hooks, etc., neither symptoms nor signs may be prominent until septic sequelæ ensue.

Case 6.—In January of last year, a boy, æt. 8 years, inhaled a small whistle. At first there was some coughing, but after a few hours all symptoms were absent. On the eighth day after the accident he was brought to the London Hospital, and the whistle was found to be in the right bronchus. Dr. Lack successfully removed the whistle by superior bronchoscopy.

Case 7.—Another case is rather famous in this hospital. In 1892 Mr. McCarthy performed tracheotomy on a man, æt. 25 years, who was suffering from stenosis of the larynx due to congenital syphilis. In 1902 the outer tube broke and a portion fell into the trachea; there were no symptoms, but he came to the hospital, and Mr. Rigby removed from the bifurcation of the trachea the portion of tube through the tracheotomy wound.

In 1904 he met with a similar accident, and Mr. Hutchinson removed the portion of tube.

In 1907 he presented himself for a third time, and on this occasion a portion of a vulcanite inner tube was removed from the bifurcation of the trachea.

Case 8.—In 1902 a victim of a similar accident was admitted to the London Hospital under Mr. Moullin. Fourteen years previously Mr. Fenwick had performed tracheotomy for syphilitic stenosis of the larynx; the outer tube had become worn, and had separated from the flange and fallen into

the trachea. Radiographic examination showed that the tube was situated about four inches below the tracheal opening. The patient was inverted and the tube recovered without difficulty.

Smooth hard bodies which are not spherical are the easiest to recover from the bronchi, but success does not always attend the surgeon's efforts.

CASE 9.—In 1894 a boy, æt. 6 years, who had inhaled a plum-stone, was admitted to the London Hospital under Mr. McCarthy. There was a violent cough and much cyanosis. Laryngotomy was performed, but as no relief resulted a low tracheotomy opening was made, and caused a slight abatement of the symptoms. Two days later the notes record that there was deficient air entry into the left lung with a rise of temperature to 103° F. The boy died on the seventh day after the accident, and at the autopsy a plum-stone was found in the left bronchus half an inch below the bifurcation; the actual cause of death was septic broncho-pneumonia.

Hard foreign bodies of irregular shape with sharp points or angles which may engage in the mucous membrane form the next group.

If the bronchus is not occluded there may be no symptoms in the earlier stages to indicate the presence of the foreign body.

CASE 10.—In 1906 an infant, æt. 3 months, was brought to the London Hospital with the history that he had inhaled a pin two days before. There were no symptoms, and physical examination of the chest revealed no abnormal signs. Radiographic examination showed a pin in the left bronchus lying with its head downwards. A week after the accident an unsuccessful attempt was made to recover the pin through a tracheotomy opening. A fortnight later, however, Mr. Rigby, working with the assistance of the X rays and a fluorescent screen, successfully removed it, and the child made a rapid recovery.

The pin had been in the bronchus nearly a month, but had caused neither symptoms nor signs of its presence.

CASE 11.—In February, 1909, Dr. Lack (22) exhibited at a meeting of the Laryngological Section of the Royal Society of Medicine a piece of bonnet-pin two inches long which had been in the right bronchus of a girl, æt. 12 years, for seven days without producing symptoms other than a slight cough. He also exhibited at the same meeting a piece of a whistle which had lodged for eight days in the bronchus of a boy, æt. 8 years, and only produced a little bronchitis (Case 6).

He had recovered both objects through a Killian's bronchoscope passed through the mouth under combined chloroform and cocaine anæsthesia.

If a body with sharp angles is not soon recovered from the bronchus, secondary troubles are likely to ensue.

CASE 12.—A man, æt. 31 years, was admitted to the London Hospital in March, 1902, saying that just before the previous Christmas he had inhaled a piece of bone. The accident was followed by severe pain in the side, which shortly subsided; a violent paroxysmal cough had persisted, and there was abundant muco-purulent expectoration. There was a subjective sensation of a foreign body near the right clavicle.

On admission to the hospital there were found dulness on percussion, weak breath-sounds, and some rhonchi at the apex of the right lung; the temperature was 100·5° F. on admission, but fell to normal three days later, and remained there. No tubercle bacilli were found in the sputum. The diagnosis was the more difficult seeing that he had been in the hospital twelve years previously under Dr. Sutton, and then had signs of pulmonary tuberculosis at the apex of the right lung. This was evidently arrested, and the man had been free of symptoms until the accident in December, 1901.

Two days after admission to hospital he coughed up a jagged piece of mutton bone, roughly oblong in shape, measuring ¾ in. by ½ in., with very sharp corners. The symptoms then rapidly subsided, though signs of scarring at the apex of the lung persisted.

The bone had been in the bronchus from December 23rd to March 6th, *i.e.* about two and a half months.

CASE 13.—In 1904 a woman, æt. 37 years, came to Dr. Head's out-patients complaining that for three months she had suffered from a severe cough with copious expectoration, wasting, and night sweating. There were then signs of bronchiectasis at the base of the right lung. She attended the out-patient department for five months; the cough became more paroxysmal, and each attack was associated with the expectoration of three or four ounces of sputum, noticed to be offensive by the

bystanders though not by the patient. She was then admitted to the hospital under Dr. Percy Kidd. The signs were those of bronchiectasis at the right base, and of broncho-pneumonia at the left base. The patient was wasted and the fingers much clubbed; eight to sixteen ounces of sputum were expectorated in each twenty-four hours; no tubercle bacilli were discovered.

The woman ascribed her illness to a chill, caused by going from a hot room into the cold air; she said nothing about the inhalation of any foreign body. There was some pyrexia (100°–102° F.) while she was in the hospital, and she died from exhaustion about nine months from the onset of the cough.

After her death her husband remarked that possibly the disappearance of an artificial tooth might have some connection with her illness. When this clue was followed, it was discovered that twice the tooth had disappeared; after the first disappearance it was recovered after its passage through the bowel; after the second disappearance it had not again been seen, but his wife's illness had commenced.

At the autopsy a tooth-plate measuring ½ in. by 1¼ in., and carrying one tooth, was found impacted in the right bronchus; there was secondary bronchiectasis of the right lower lobe, and septic pneumonia on the left side. The specimen is on the table.

This case is remarkable in that a large foreign body inhaled and lodging in a bronchus apparently produced no symptoms until secondary bronchitis and bronchiectasis ensued; the reticence of the woman and her husband prevented the discovery of the clue to the real diagnosis.

In the following case there was a similar absence of early symptoms.

In September, 1905, a lady was sent to see me by Dr. Graham, of Wonersh, who complained of a cough with copious expectoration, which began as a cold in November, 1904, and had persisted since that time; from January 7th, 1905, the sputum had been offensive.

There were no other points of importance in the personal or family history except that on putting a leading question I elicited the fact that in October, 1903, thirteen months before the onset of the cough, she had had four teeth removed under gas; all went well after the operation except that she could "taste the gas" for two or three months. On inquiry the dentist said that he was positive that all the teeth removed had been seen after the operation.

On physical examination I found slight pleuritic friction and some weakening of the breath-sounds in the middle of the right axilla. Beyond these points I could not detect any abnormality. The sputum was typical of bronchiectasis. Examination with the X rays failed to reveal any foreign body, but showed a shadow in the periphery of the right lung. Treatment by creasote baths was practised without any appreciable benefit in the symptoms of bronchiectasis. The general nutrition was well maintained. On April 4th, 1906, *i.e.* two and a half years after the dental operation, she was cycling from Guildford to London, and while struggling up a rather stiff hill she had a violent fit of coughing and ejected the stump of a tooth with two fangs attached. Soon after this the cough and expectoration disappeared, and now it is only when she gets a cold that she occasionally brings up a little sputum in the morning to remind her of her old trouble.

The result of this case was satisfactory, though I fear rather by good fortune than by good management. At the present time a similar case should certainly be investigated with the assistance of the bronchoscope.

Foreign bodies which are not hard may swell under the influence of moisture after inhalation. Killian points out that the importance of this group lies in the difficulty of removal, and recommends inferior bronchoscopy after a preliminary tracheotomy. Vegetable substances, such as maize leaf, peas, and beans, fall into this category, but I am unable to quote to you a case in which the swelling of the body after inhalation was proved.

CASE 14.—It is possible that swelling prevented the ejection of a pea in the following case. In 1898 a woman, æt. 28 years, had a short attack of pleurisy. A year later she was admitted to the London Hospital under the late Sir Stephen Mackenzie with signs of left basal bronchiectasis, with pyrexia due to intercurrent pneumonia. Shortly after admission she began to cough up large quantities of foul sputum. Under treatment by creasote baths she improved, and was discharged after six weeks still with signs of bronchiectasis and free foul sputum. She continued in moderate

health for nine years, the cough persisting and the sputum being occasionally offensive. In 1908 she was re-admitted under Dr. Hadley five days after a rigor; the following day she coughed up a pint of foul pus. A portion of the eighth left rib was then resected, and the finger inserted into a cavity in the lung from which a small foreign body the size of a pea was extracted. On microscopical examination the foreign body was found to be of vegetable origin.

The woman died of exhaustion, and a post-mortem examination was not permitted.

It seems possible that the pea when first inhaled was not coughed up because it swelled up and so became impacted in the bronchus.

Soft foreign bodies which do not swell under the influence of moisture are found fairly frequently.

Case 15.—Last year a boy, æt. 1½ years, was admitted to the Hospital having inhaled some pieces of almond. Superior bronchoscopy was performed and two fragments of almond were removed, one from the trachea and one from the left bronchus. Unfortunately the child died two days later from acute œdema of the larynx and septic broncho-pneumonia. Tracheotomy relieved the secondary obstruction but did not save life.

Case 16.—Another child, æt. 13 months, was brought to the Hospital in 1903 almost moribund, having inhaled an orange-pip. Tracheotomy was immediately performed, and the pip was coughed up through the wound. Ten days later the child died from secondary broncho-pneumonia.

Case 17.—A case with a happier termination occurred in 1908. A girl, æt. 5 years, was brought to the Hospital with a history of having "swallowed a marble." There was much stridor and consequently tracheotomy was immediately performed. The foreign body was not found. A few hours later the dyspnœa recurred and a foreign body could be felt hitting the tracheotomy tube. Inversion of the child with shaking failed to remove it. The trachea was then stitched to the skin and search was made with forceps. A castor-oil bean was removed. Broncho-pneumonia ensued and the temperature rose to 105° F., but after ten days convalescence was established and the child was discharged perfectly well.

In America the commonest soft foreign body inhaled into the air-passages seems to be a cockle burr. In London, of course, such an accident is uncommon.

Mr. Godlee has pointed out that many cases of bronchiectasis seem to be caused by inhalation of pieces of adenoid tissue or tonsil during the anæsthesia induced to facilitate their removal.

A special group of soft foreign bodies not affected by moisture is worthy of attention. It consists of those bodies such as ears of grass, which by their peculiar conformation tend to migrate as a result of movement.

I have already alluded to Sir Thomas Watson's (16) case in which an ear of rye worked its way from the lung into the liver of a coachman who inhaled it. It has been suggested that this case was really an example of pulmonary and hepatic actinomycosis. There are now on record several examples in which the means of infection by the actinomyces have been proved to be an ear or a grain of some cereal.

In one of Mr. Godlee's cases (17) a head of grass worked its way through the sixth left intercostal space three inches from the spine forty-three days after inhalation.

The secondary effects of an inhaled foreign body, whatever its physical characters, will depend largely on the nature of the micro-organisms it bears with it. It is unlikely that it can ever be quite sterile.

Symptoms and signs.—I have detailed many examples of the different effects which may ensue upon the inhalation of a foreign body into the air-passages.

Both symptoms and signs, primary and secondary, vary with the point of lodgment of the foreign body and upon its physical characteristics. If the foreign body lodges in the undivided air-way between the glottis and the bifurcation of the trachea, the immediate result may be sudden death from asphyxia; if the obstruction is not complete there may be more or less cough and dyspnœa, and in some cases the patient may remain unconscious of the presence of the foreign body.

The remote or secondary consequences also vary with the situation and physical characteristics of the foreign body; œdema of the larynx, ulceration of the mucous membrane with subsequent stenosis, and septic broncho-pneumonia are the commoner complications, and of these I have already quoted examples.

If the foreign body passes into one of the

bronchi, both symptoms and signs, immediate and remote, depend chiefly upon its physical qualities. In many cases, of which I have quoted examples, the immediate effects after the initial fit of coughing are scarcely noticeable; in other cases, a cough, paroxysmal or continuous, persists from the time of the accident until the foreign body is rejected.

The secondary effects of a body allowed to remain in a bronchus are varied; they may be purely mechanical, *e.g.* the perforation of an important organ by the migration of a sharp-pointed foreign body, and the collapse of an area of lung owing to occlusion of its bronchus, or an infective process may be superadded to the mechanical effects. In this way there may result: septic bronchitis, ulceration of bronchi, hæmoptysis, broncho-pneumonia, bronchiectasis, pulmonary abscess or gangrene, empyema, perforation of the chest-wall with the establishment of a bronchial fistula, actinomycosis.

Diagnosis.—When the history of the accident is provided, the diagnosis of a foreign body in the air-passages is an easy matter, though occasionally it may be difficult to decide at once whether the foreign body was passed into the air-passages or down the œsophagus.

Foreign bodies in the œsophagus.—Inspection of the pharynx and digital exploration are of very great importance; the passage of an œsophageal bougie may at times be of service, but is not without danger if the foreign body impacted in the œsophagus be hard and sharp. If time and circumstances permit, it is safer to examine with the X rays and with the œsophagoscope. When using the X-ray screen remember that flat bodies, such as coins, in the œsophagus, lie in the transverse diameter of the body, whereas if in the trachea they may lie in other positions. The trachea and bronchi move downward on inspiration and upward on deglutition; these points may be of assistance in diagnosis when using the X-ray screen. Unfortunately many foreign bodies are translucent to the X rays and therefore are not demonstrable by their aid.

If the history of the accident is not forthcoming the diagnosis may be very difficult. I have quoted examples to show how it has been the cause of sudden death in cases where apoplexy, epilepsy, alcoholism, etc., were assumed to be the determining factor.

Laryngeal obstruction of other origin.—The laryngeal obstruction due to a foreign body has been ascribed to laryngismus stridulus, œdema of the larynx, diphtheria, etc. In all cases where the obstruction has had a sudden onset the possible presence of a foreign body should be carefully considered.

Bronchiectasis; abscess of lung; empyema.—When secondary changes in the lungs have occurred the diagnosis from bronchiectasis, abscess of lung, or empyema due to other causes may present great difficulty; we should never be content with the simple diagnosis of bronchiectasis or abscess; we should invariably use every endeavour to trace the ætiology.

The assistance of a skilled radiographer is of very great value, if the foreign body is opaque to the X rays.

When there is an empyema due to a foreign body, in the absence of any history of its inhalation it is impossible in most, if not all, cases to determine its presence before the operation. Fortunately the treatment is the same whether we are certain or uncertain as to the cause.

Pulmonary tuberculosis.—The presence of a foreign body in a bronchus has often in the past led to a diagnosis of pulmonary tuberculosis. With modern aids to the diagnosis of pulmonary tuberculosis this mistake should seldom occur; the combination of the two conditions, as in one of the cases I have quoted, may enhance the difficulty very greatly. The development of the tuberculosis may be antecedent or subsequent to the accident.

Pertussis.—The paroxysmal cough due to the presence of a foreign body in the air-passages may suggest the diagnosis of whooping-cough; in mos cases careful investigation serves to establish the diagnosis.

Finally I would urge you never to discuss lightly, as of no importance, the patient's story of the inhalation of a foreign body. Moreover it is wise to inquire carefully for such history if there are signs of pulmonary disease of obscure origin at the base of one of the lungs, especially on the right side, because, owing to the slight symptoms in the early period after the accident, the patient often fails to attach any importance to it. Especially is this the case if the patient was unconscious when the foreign body was inhaled.

Prognosis.—The prognosis after the inhalation of a foreign body into the air-passages depends on so many circumstances that it is not possible to lay down any definite laws. The body may be removed naturally or artificially in a short period of time, and no secondary effects may ensue.

Secondary effects in the lungs may develop and disappear after the removal of the foreign body, or they may persist in spite of its removal.

Bronchiectasis or abscess of lung may lead to abscess of the brain or lardaceous disease or other well-known sequelæ.

Empyemata and bronchial fistulæ may also give rise to permanent trouble.

Migration of a foreign body from its first resting-place may cause serious complications.

These instances are sufficient to show that the prognosis in each individual case requires separate consideration.

Treatment.—The treatment of foreign bodies

inhaled into the air-passages is usually considered to fall within the province of the surgeon. At the time of the accident, however, the nearest doctor will be called upon to act promptly and quite independently of professional specialism. In the later stages, when complications have developed, the course of treatment is best determined by the physician and surgeon in consultation.

I may be pardoned, therefore, for presenting to you a brief critical review of the methods of treatment. At the time of the accident there is generally a fairly violent, and often successful, natural effort to expel the body by coughing, not unusually assisted by the bystanders by a thumping on the back; theoretically, this thumping should be synchronous with, and intended to aid, the expiratory effort; practically, I fear it is often performed without any clear idea of the nature of the assistance intended. Should these preliminary efforts be ineffectual, the pharynx should be explored with the finger in case the body should be lodged within reach, but care must be exercised lest it should be pushed further down. If not removed thus, the next step must be determined by the urgency of the symptoms. If asphyxia seems imminent, the undivided air-way is probably blocked at its narrowest part, *i. e.* the glottis. Laryngotomy or tracheotomy with any available instrument is necessary; inversion and shaking should also be practised. Hang the patient over your back with his knees flexed over your shoulders and shake him; two operators are better than one if the patient is heavy. The degree of asphyxia will determine whether the inversion should be attempted before or after opening the trachea.

If the symptoms are less urgent, time may permit a more orderly procedure. In the old days sternutatories like pepper or snuff, or emetics such as mustard and water, were administered; these seem to have been uniformly useless and often harmful.

In the past there has been a good deal of dispute as to the value of inversion in the treatment of this accident. In the case of Brunel it was found that when the half-sovereign fell back against the under-surface of the larynx it caused so much spasm that he was nearly asphyxiated, and the attempt had to be abandoned until a preliminary tracheotomy had been performed. It is noteworthy, however, that the coin was ultimately recovered through the mouth and not through the tracheotomy wound.

Modern teaching seems to be that inversion should never be practised in this class of case until tracheotomy has been performed. A study of the cases seems to show that provided everything is ready for immediate tracheotomy in case of spasm of the larynx, no real harm can result from the inversion of a patient who has inhaled a smooth, hard body, spherical or irregular, such as a pebble, nut, collar-stud, button, coin, etc., but that the attempt should not be made if the body is pointed or jagged, such as a tooth-plate, a pin, a nut-shell, a piece of bone, etc., or not hard, such as a piece of meat, an ear of corn, a piece of tonsil or adenoid growth, etc.

In these two groups the choice of procedure will be between tracheotomy and inversion, tracheotomy and search with forceps, tracheotomy and inferior bronchoscopy, and superior bronchoscopy.

The first two methods are comparatively simple, and may be performed by any doctor who has the necessary instruments. The last two methods require a knowledge of the use of the bronchoscope, an instrument which few as yet have employed sufficiently often to have become proficient. The bronchoscopic method, however, has several great advantages; the operator can see what he is doing, and when the instrument is passed through the mouth a cutting operation is avoided. Brilliant success has attended the attempt to remove a foreign body with forceps through a tracheotomy wound with the assistance of an X-ray screen; but it is obvious that this method is useless unless the body is opaque to the rays, and at the best the operator cannot actually see the object he is attempting to remove.

Bronchoscopy, on the other hand, has certain limitations apart from the necessity of waiting for a skilled operator.

If the foreign body has led to hæmorrhage, to inflammation or ulceration, the presence of blood, mucus, or pus may so obscure the view that it is impossible to see the foreign body. In this case it scarcely presents any advantages over the older method of groping with forceps through a tracheotomy wound, guided by the sense of touch or by the shadow on the fluorescent screen. There is just a chance that superior bronchoscopy may be effectual, and in this case the tracheotomy wound would be avoided.

Another disadvantage of superior bronchoscopy is that the passage of the tube through the small larynx of a young child may cause inflammatory œdema and laryngeal obstruction sufficient to call for subsequent tracheotomy.

To sum up, the bronchoscopic method by the superior route seems to be the best, provided that time permits, when irregular, hard, jagged, or pointed objects have been lodged for but a short time in the trachea.

If the object be not hard, or if it has been in the air-passages long enough to produce secondary changes, then bronchoscopy through a tracheotomy wound should be recommended. If the body has penetrated into the lung substance and led to pulmonary abscess and to an empyema, it is necessary to approach the seat of disease through the chest-wall, but the operation should cease with the opening of the cavity and the evacuation of its contents.

Attempts to reach a bronchus and to remove therefrom a foreign body by the trans-thoracic route seem to have been uniformly unsuccessful and often fatal, and should not be recommended. Recently attempts (26) have been made to diminish the danger of this operation by enclosing the patient's head in a box and administering air and chloroform under pressure so that the lung is kept distended when the thorax is opened; or the process is reversed—the patient's body up to the neck is placed in a box in which the atmospheric pressure is reduced below the pressure of the air breathed by the patient through the mouth.

Mr. Godlee has pointed out that the advantage of either method must immediately cease if either the lung tissue or the bronchus is wounded.

Finally I would warn you that though the prognosis is grave if an inhaled foreign body be not removed from a bronchus, yet it is not necessarily fatal. I have already quoted to you a case in which a tooth was coughed up two and a half years after its inhalation with subsequent disappearance of symptoms.

At the end of the lecture I will show to you a boy who inhaled a piece of filbert shell in June, 1907. Symptoms of bronchiectasis ensued, and he was sent to see me at the Brompton Hospital by Dr. Ward, of Grays. In September, three months after the accident, he coughed up the nut-shell, and speedily lost all his symptoms.

Case 18.—Sometimes the body is coughed up but the secondary bronchiectasis persists. I am able to show you to-day a woman who in 1906 inhaled a piece of mutton-bone as large as a tooth; she developed symptoms and signs of bronchiectasis, and in March, 1907, coughed up the piece of bone. Dr. Arthur was in attendance, and, finding that the symptoms persisted, he sent her into the London Hospital under the care of Dr. Dawson.

There was then evidence of right basal bronchiectasis and some suspicion of an abscess cavity in the lung. At Dr. Dawson's request Mr. Openshaw resected a portion of the ninth right rib and explored the lung. No abnormality could be detected, and consequently the wound was closed. The patient's condition was unchanged by the operation. The bronchiectasis has persisted, and she is now an inmate at St. Joseph's Hospice, whence, by the kindness of Dr. McNaboe, she has been permitted to come here to-day.

Case 19.—Dr. Lack and Dr. Hann (23) showed a case before the Royal Society of Medicine in October, 1908, in which a tooth-stump had been inhaled in February of the same year, when the patient was under an anæsthetic; three weeks after the accident purulent expectoration began, and in June there were definite signs and symptoms of left basal bronchiectasis. In September, 1908, and again, in November, Dr. Lack attempted to recover the tooth through the bronchoscope, but was baffled by the amount of secretion.

Dr. Hann has kindly written to me this week, and reported that the man is still at his work, and has scarcely any signs or symptoms.

Case 20.—In January, 1908, there was a man in this hospital under my care who for two years had had a piece of mutton-bone in his left bronchus; there were symptoms and signs of left basal bronchiectasis, and the bone was seen with the help of the X rays. Dr. Lack very kindly attempted the removal through the bronchoscope, and though the bone was seen and felt, yet, owing to the free secretion, removal was not possible without undue risk to the patient's life. The patient left the hospital in much the same condition as when he entered it. I have not been able to obtain any news of his subsequent history. I think that he has emigrated.

An example of death from cerebral abscess in a case of bronchiectasis secondary to the impaction of a piece of bone in a bronchus is recorded by Bamberger, and quoted by the late Dr. Schorstein in his thesis on cerebral abscess in connection with pulmonary disease (24).

Mr. Godlee (25) has recorded a case of a boy who, at the age of nine, in 1885 inhaled the iron peg of a peg-top; in 1893 he was admitted to University College Hospital with signs and symptoms of left basal bronchiectasis. Mr. Godlee resected a portion of rib, stitched the lung to the chest-wall, opened a bronchiectatic cavity, and searched diligently, without success, for the foreign body.

The boy went home, and eight months later the iron peg, in a fit of coughing, was discharged through the fistula; he began to improve very gradually. Dr. Hopkyn Davies wrote this spring to say that the patient enjoys excellent health, is married, and has two children. He has a slight morning cough with some expectoration, and the fistula still discharges. The history is thus carried on for twenty-five years.

In another of Mr. Godlee's (25) cases the history is carried on for nineteen years. In February, 1891, a boy, æt. 6½ years, inhaled an ivory knob; eight weeks later he ejected it, but left basal bronchiectasis had been established and still persists (*i. e.* in 1910). He still has to empty his bronchiectatic cavities regularly by stooping forward, but except for this he is very little incommoded by his trouble.

I have quoted these cases to show that it is desirable to use every effort to remove an inhaled foreign body, though if it is removed complete cure does not necessarily follow, and if it is not removed a rapidly fatal termination does not always ensue.

I have not detailed all the methods that may be employed in dealing with this accident; the ingenuity of the surgeon will often suggest a con-

venient method at the time of the operation. Thus Barclay (27), of St. Louis, had to remove from the larynx of a patient a cockle burr which was lying between the vocal cords with the sharp spine at the tip embedded anteriorly just above the commissure of the cords.

After cocainisation Barclay caught the burr with forceps, and told the patient to continue phonating *'aa"* as long as possible; when desperate for air he permitted her to inspire and at the same time pulled out the burr entire; in this way he avoided the laryngeal spasm.

CASE 21.—In 1897 Sir Frederick Treves had to deal with the case of a small boy, æt 20 months, who was admitted to the London Hospital with a dress-hook lodged in the ventricle of the larynx; this could be felt with the finger, but could not be removed through the mouth.

Sir Frederick Treves performed thyrotomy and removed the hook with forceps. The child recovered rapidly and the voice was natural in fourteen days.

I think that I have sufficiently indicated that common sense rather than rigid rules should guide the action of the doctor who has to deal with the accident which has formed the subject of to-day's lecture.

Before deciding upon the mode of treatment it is wise to take into consideration the nature of the foreign body, the age and condition of the patient, the lapse of time since the accident, and the complications which may have ensued. An attempt to "reconstruct" the accident may throw much light upon the position in which the body lies, and thus assist the operator in his attempts at its removal.

In conclusion, I desire to express my thanks to my colleagues for their courtesy in permitting me to make use of the cases under their care.

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June 20th, 1910.

THE TREATMENT OF INDIGESTION.*

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I HAVE been asked to speak this evening on some subject of daily importance to us all as practitioners of medicine, and have, not, I think, without courage, chosen that of the treatment of indigestion. Nearly every doctor has his own plan or plans for dealing with the stomach-ache, and as many forms of the affection are curable by simple means, can claim a large measure of success. We have all, however, to acknowledge our defeats, and though these are often due to lack of co-operation on the part of the patient, it may be of advantage to us to discuss the common means of treating indigestion and the forms of the disorder to which they can be most properly applied.

The term indigestion really includes difficulty or failure of digestion in any part of the alimentary tract, but is often used of the stomach only, and in that colloquial sense I use it this evening. Indeed, apart from certain severe diseases, we know very little of indigestion in the small intestine, though plenty of printed statements on the subject reach us by the post, particularly accompanying samples of ferments. No part of the human frame, and no function, is exempt from the seller of quack medicines and the writer of advertisements; but his gifts are seen to the greatest advantage when he is dealing with disorders, real or supposed, of which little or no exact knowledge exists.

Indigestion is often secondary to general diseases, such as the anæmias, Addison's disease, diabetes, and many others; or to diseases of other organs such as the heart, lungs, liver, kidneys; or, again, to gross affections of the stomach and intestines, as growths, ulcers, stenosis of the pylorus, or intestinal obstruction. It is assumed that in all cases a thorough examination of the patient has been made to ascertain whether the indigestion is a complication of some more serious condition, or whether its origin can be regarded as in the stomach itself. Of course a secondary indigestion may require to be treated just as much as the primary form, and often by the same methods, but success is not likely to be attained unless attention is also given to the original disorder.

Indigestion may be caused by a number of different diseased states of the stomach involving the secretion of gastric juice, the mixing of the food in the stomach, and its discharge into the duodenum. We cannot pretend that our treatment is likely to be sound unless the proper steps are taken to find out which of these functions of the stomach is in abeyance or is exaggerated, and to what degree. Can we determine by questioning the patient and by examination of the abdomen whether he is the subject of gastric catarrh, of hypersecretion, of achylia gastrica, of delayed disharge from the stomach due to atony, or to obstructive dilatation? Or if a diagnosis of gastric catarrh is made, can we by these means determine whether the hydrochloric acid and the ferments are present in a natural proportion or not? It must be admitted that this question cannot be answered in the affirmative, for an accurate diagnosis can only be made by an examination of the contents of the stomach. These are often furnished by the act of vomiting, and should be examined microscopically and chemically. If there is no vomiting, then some material may be obtained by means of the stomach-tube. In a great many cases we cannot be sure that the right treatment is being prescribed unless such an examination has been made.

The stomach-tube is not, however, to be lightly used on all and sundry. To some it is harmful, even dangerous, to others it is unnecessary, and to all unpleasant. We may, therefore, consider what steps may be taken without the use of the test-meal and the tube. The first step is to try and find out the particular stress which has caused the onset of dyspepsia. Obviously the only rational method of procedure to begin with is to treat the dyspepsia by removing its cause, if that is possible. The chief cause of dyspepsia is overwork. The establishment of regular habits of work, rest, and recreation will put many people right without any other measures being taken. A close ally of overwork is the habit of irregular meals eaten hastily. The teeth must be carefully examined from two points of view—first, to see that there are enough sound teeth, and in a suitable position, to masticate the

* Read before the Harveian Society of London.

food; and secondly, to see that there are no decaying stumps or pyorrhœa alveolaris, which may discharge fœtid matter likely to set up a chronic infective gastritis. The tongue must also be examined, and an antiseptic mouth-wash ordered to be used regularly if there is any septic focus in the mouth at all. Dental defects are a common cause of dyspepsia, but the fact that they are much commoner than even dyspepsia is, shows what burdens the healthy stomach will endure. I had for some years under my care a woman, who, when a child, had had an abscess involving the right temporo-maxillary joint, resulting in complete ankylosis. The teeth were fixed firmly together, the food being introduced through a square hole of fair size made by the extraction of incisors. She ate everything her family ate, chewed nothing, of course, did not mince her meat, but put in any piece the aperture would admit, and though she was past middle age, had never had indigestion.

Errors of diet are a common cause of dyspepsia, though seldom the only cause. An indiscretion in the direction of over-feeding is often the cause of an acute gastritis which may become chronic. Alcohol is, however, far more often than unsuitable food, the cause of dyspepsia. Tobacco is well known as a contributory cause, and especially cigarettes; not only cigarettes, however, for any form of tobacco-smoking in excess may be harmful. Both with alcohol and tobacco abstinence is better than restriction, at all events for a time; it is a more sound test for one thing, and for another, mere restriction is apt to be interpreted too leniently by the patient. Abstinence, by the doctor's advice, everybody understands. But, in the case of habits of years, patients do not understand what moderation is. A gentleman who had travelled widely, when asked about smoking, said he did not refill his cigarette case more than once a day. I asked after the case, and he produced a box like a breastplate holding about twenty cigarettes each side.

Exposure to cold is another factor in the causation of indigestion, as many dyspeptics can testify. The feet and the abdomen should be kept warm; the wearing of a band of woollen material round the abdomen cannot be necessary in warm weather, though it may be in cold. It is probably better to wear a thick flannel shirt in the winter and a thin one in the summer than to increase the apparatus of life by the marvellous varieties of belts which we see among out-patients. The habit of wearing knitted socks or stockings in the winter, which appears to have gone out to some extent, is, I think, a good one, especially for those who are much out-of-doors and who travel about in trains, trams, buses and motor cars. Anxiety, worry, and the neurasthenia they bring about are also frequent causes of dyspepsia. The most valuable feature enabling us to distinguish nervous dyspepsia is perhaps the variability of the symptoms in relation to annoyances, but irrespective of diet.

A large number of cases of indigestion may be cured by attention to the above points. If the doctor is of opinion that the cause of the disorder lies in one of these factors, it is important that the patient should recognise that his cure depends upon himself and not upon a bottle of medicine.

Certain forms of indigestion may be commonly recognised from the symptoms without any examination of vomited matter or the material obtained by the stomach-tube. I do not refer here to the dyspepsia of chlorosis, as that is included in the secondary dyspepsias mentioned in my third paragraph: neither am I dealing with acute gastritis.

Hypersecretion of gastric juice causes a characteristic series of symptoms, especially pain or discomfort coming on some time after food, the interval being longer when protein is taken in quantity, and shorter when starches or sugars are taken; eructation, pyrosis, hunger, and the occasional vomiting of a large amount of acid fluid, with perhaps alkaline urine. These symptoms are only to be confounded with those of duodenal ulcer, and doubtless some cases of ulcer have been regarded as severe cases of hypersecretion or hyperacidity as it has been called. These symptoms are by no means infrequent, and they yield very easily to appropriate treatment. Meals should consist of solid food only and be given every three or four hours, with at least a quarter of a pound of meat at chief meals, and plenty of butter. Sodium bicarbonate is ordered in half-drachm doses or more in a medicine when the pain comes on Fluids are drunk an hour before meals.

Another condition which can be easily identified in most cases is that of dilatation of the stomach leading to stagnation of its contents. The pain at night, the account of the vomiting of a large quantity of material consisting of the food of

several meals, the thirst, and the examination of the abdomen, confirmed if necessary by the X rays, are distinctive. In its well-marked forms this condition is due to pyloric stenosis, and its proper treatment is gastro-enterostomy. It is true that washing out the stomach will give such enormous relief that the patient may regard himself as cured. This may be in itself, however, a danger, for I have known a patient refuse operation on that account, and return for treatment months later when the chance of the successful excision of a pyloric growth was gone.

The majority of those who seek advice do not present such a well-defined series of signs, but complain of one or more of the symptoms known as dyspeptic. A consideration of the results obtained from the many examinations of the gastric contents that have now been made shows us that we cannot diagnose with certainty from those symptoms whether there is a chronic inflammatory condition of the stomach or not, and whether the secretions are altered or absent. We may classify the cases from the symptoms to our hearts' content, but though we may often be right, we shall often be wrong. Nevertheless, a great number of these patients can be cured without using a tube, even when no vomited matter is available, by applying the results of past experience, and that being so, if a patient has not been treated, my opinion is that the simple means we all know of should first be used. I prefer very much to make an accurate diagnosis, especially when it can be done by so easy a procedure as a test meal; but that does not justify me in passing a tube in every case of dyspepsia when I know that there is a good probability of relieving the malady without that operation.

Of 425 cases of dyspepsia under my care of which I have taken notes, 61 were well-marked cases of hypersecretion, dilatation, alcoholism, or the dyspepsia of young children. The remaining 364 complained of pain or discomfort shortly after food, with such symptoms as the following: burning and smarting in the stomach and back, in many cases worse on exertion, or in cold weather; distension; sinking, faintness, a feeling of emptiness, specks before the eyes; palpitation, choking in the throat, dyspnœa; spasms, flatulence, eructations, pyrosis, retching, vomiting of "phlegm" or food; loss of appetite; tiredness; constipation, diarrhœa.

I have looked through the treatment given to all these patients and find that the majority of them were successfully treated by advice as to habits, particularly with reference to rest, alcohol, and tobacco, as to diet, and by certain simple medicines. In order to draw up rules of diet the daily food of the patient must be inquired into. Pastry, potatoes, greens, new bread, alcohol, and tea should be avoided, and fluid taken at the end of a meal, or in severe cases an hour before the meal. On looking through the many medicines used, I find that by far the most useful were alkalies, carminatives, bismuth, and bitters. Sodium bicarbonate is given an hour to an hour and a half after a meal or when pain and discomfort comes on. It arrests the continuous weak secretion in gastric catarrh, as Pawlow showed, thus sparing the glands to produce a better fluid at the next meal; it helps to dissolve the slimy material produced by the mucous glands; and it neutralises acid if that be present in excess. A carminative in the medicine stimulates the stomach to discharge the air swallowed in nausea and any gas formed in the stomach, and increases the blood-supply of the stomach-wall. The following is the most generally useful prescription of those I have given: Bicarb. of soda 25 grains, tr. of ginger 30 drops, chloroform water to an ounce. Bismuth is also very valuable, especially when hyperæsthesia is present; it is usually given before meals and in good quantity, half a drachm at least of the carbonate or oxychloride, as a powder, or shaken up in water with no mucilage. If the bismuth is suspended by mucilage in the water, it will be so in the stomach. The liquor bismuthi et ammonii citratis, which throws down its bismuth when exposed to the acid reaction of the stomach, embodies a good idea, but there is nothing like enough bismuth in it—only about three grains in each drachm. If the appetite is poor and the juices are regarded as deficient, then bitters are given before meals in acid solution.

These time-honoured measures, and others like them, are, as I have said, successful in the majority of ordinary cases. There remains, however, a large number of cases which do not yield in a trial extending over two or three weeks or longer. If an accurate diagnosis has not been made from vomited matter, the patient should then be given a meal of three or four ounces of bread and half a pint of weak tea in the morning and some of his stomach

contents obtained by the tube an hour or so later. If a large amount of mucus is present and is not derived from the saliva (which contains small bubbles), he is the subject of gastric catarrh. If microscopical examination shows glandular cells and leucocytes in excess, then chronic inflammation is present, whether there be an excess of mucus or not. In chronic gastric catarrh the percentage of hydrochloric acid may be increased, normal or diminished, or the acid may be absent. If it is diminished in quantity or absent the ferments are usually deficient too.

The treatment of chronic gastric catarrh will be guided by the results of the examination. The evidence of subacute inflammation of the mucous membrane indicates that fluid food should be given, unless there is excellent masticatory power, in which case a little solid food may be allowed. The bulk of the dietary must, however, consist of milk with cream, butter, and eggs. Very hot and very cold food and drinks must be avoided. If the hydrochloric acid is in normal proportion or in excess, protein may be ordered more freely, such as eggs, tender chicken or mutton, and fish. If the acid is diminished or absent flavoured foods and soups may be given to arouse a secretion, but, of course, no peppers or irritating spices. Meats must be minced or very tender, as connective tissue will be badly digested in the absence of an efficient gastric juice. The stomach must not be exposed to the irritation of metallic or vegetable purgatives, or of magnesium sulphate. Water may be taken fasting for constipation, and enemas should be used if required. If the amount of mucus is excessive and remains so in spite of treatment, the stomach may be washed out daily for a time with a weak alkaline solution.

If there is no excess of mucus in the material obtained and microscopical evidence of inflammatory exudation is wanting, whilst the acid is absent or diminished and the test-meal undigested, the condition is one of simple deficiency of secretion. Spicy foods should be ordered to be taken chopped up small or semi-fluid at first, but not much meat or protein. The administration of ferments and even of gastric juice itself has not justified the hopes which were at one time aroused. A medicine containing hydrochloric acid may be given, with iron if needed, and general "tonic" treatment. A stay at a spa is often beneficial, with hydrotherapy, especially in cases with a large neurasthenic element.

The treatment of hypersecretion has already been outlined.

If motor deficiency is suspected, the stomach should be washed out three or four hours after the test-meal, or better, a test supper of bread, meat, and water or cocoa given at night, and the stomach washed out in the morning. If there is any residue in the stomach the diagnosis is confirmed; it may also be made by repeated X-ray examinations after a meal of bread and milk or porridge mixed with bismuth carbonate or oxychloride.

Weakness of the abdominal walls must then be treated by exercises and massage, with the use of a belt or binder for a time when in the upright position. The meals should be small, and fluid taken between but not at them, and restricted to two to three pints a day. Strychnine, and, if necessary, iron, may be given. The character of the food ordered will be determined by the amount of hydrochloric acid and the presence or absence of ferments upon the lines already detailed.

If the material obtained from the stomach is of a sour smell, and consists in part of the food of earlier meals, and contains sarcinæ and yeast-cells, then actual stagnation of food has taken place. Such a condition is, as above mentioned, enormously relieved by washing out the stomach, with the dietetic measures referred to in the last paragraph, and the use of sulpho-carbolate of soda or salicylic acid. If the urine is scanty, fluid may be given *per rectum*. But, except in certain uncommon circumstances, the stagnation of food in a dilated stomach is due to gross obstruction, and should be treated surgically.

I cannot, gentlemen, go into more detail without encroaching upon the time allotted, but I hope that enough has been said to show with how much greater accuracy and likelihood of success indigestion may be treated when a knowledge is obtained of the actual conditions in the stomach, by an examination of its contents.

June 20th, 1910.

We have received from Messrs. Burroughs Wellcome & Co. specimens of Tubercle Vaccine and New Tuberculin, human and bovine, for report.

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CONTENTS.

PAGE

*A Clinical Lecture on Inguinal Ruptures which are difficult to manage; and their Treatment. By C. B. LOCKWOOD, F.R.C.S. 177

*Syphilis. A discussion by Mr. ERNEST LANE, Mr. ALEXANDER FLEMING, Major H. C. FRENCH, R.A.M.C., Mr. J. E. R. MCDONAGH, Mr. C. F. MARSHALL, Dr. PERNET, Mr. H. W. BAYLY 181

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A CLINICAL LECTURE

ON

INGUINAL RUPTURES WHICH ARE DIFFICULT TO MANAGE; AND THEIR TREATMENT.

Delivered at St. Bartholomew's Hospital.

By C. B. LOCKWOOD, F.R.C.S.,
Surgeon to the Hospital.

GENTLEMEN,—I propose to lecture to-day upon inguinal ruptures which are difficult to manage, and their treatment. Most of your patients with inguinal ruptures are young people who will have radical cure done. But there will be a residuum of older people who elect to be treated with trusses, and in these difficulties may arise At times you will be puzzled by the early stages of inguinal hernia and the clinical signs may be difficult to interpret. One who feels a pain in the lower part of the right side of the abdomen is almost certain nowadays to think that he is suffering from appendicitis, although the pain is caused by the commencement of a rupture. You will, however, be put upon your guard when told that the pain is absent during the early part of the day, and thus it comes on towards evening, after standing or exercise. Under such circumstances proceed as follows: The patient being stripped, cause him to stand up in a strong light, then ask him to cough. Keep the eyes fixed upon the situation of the internal abdominal ring, and if there is a localised impulse it will be seen. Some impulses are more easily seen than felt.

Next, lay the patient down, and invert the scrotum with the index finger and thrust its tip into the external abdominal ring. The normal external abdominal ring should just hold the tip of the finger, its edges should feel sharp, and every time the patient coughs or strains the tip of the finger

should be extruded. If the external abdominal ring admits the finger and is loose and does not contract, your suspicion should be awakened, especially if you have seen an impulse.

Next, if the first joint of the index finger can be passed through the external ring into the inguinal canal you may consider that the canal is defective, and if when the patient coughs or strains you feel an impulse against the tip of your finger, the probabilities are very strong that, when the inguinal canal is opened a commencing hernia protrusion will be found. Whilst the finger is in the inguinal canal two other points should be cleared up. I now have my finger in the right inguinal canal of this patient. When I turn the pulp of my finger backwards towards the abdomen I find that the posterior wall of his inguinal canal is firm and strong, and prevents my finger entering the abdomen. Now I ask him to cough—I do not feel the arching fibres of the internal oblique and transversalis muscles contract, descend, and close the canal. As you are aware, the arching fibres of the internal oblique and transversalis muscles are so arranged that when they contract they close the inguinal canal. This young man is anxious to have a radical cure done. We may infer that when this is undertaken we shall find the inguinal canal has a good back to it, but that the arching muscular fibres are weak and thin, and not very good material to work upon. When the hernial protrusion has passed through the external abdominal ring and become complete, difficulties in diagnosis hardly exist. Ruptures which are not treated either by operation or by trusses are almost certain to increase in bulk and may attain unmanageable dimensions. I can recall the case of a man whose inguinal rupture had attained such dimensions that he was unable to follow his business and suffered from continual pain and occasional attacks of obstruction. When the sac was opened it contained $2\frac{3}{4}$ lb. of omentum (which was removed), part of the transverse colon, many coils of small intestine with their mesentery, the cæcum, the vermiform appendix, and part of the right colon.

A neglected rupture also causes changes in the inguinal canal which it is necessary for you to understand. As I have already pointed out, in the early stages the internal opening at the neck of the sac of an oblique inguinal hernia is situated at the outer end of the inguinal canal. The finger placed upon the abdominal wall just inside and above the centre of Poupart's ligament marks this internal opening. Perhaps I ought to speak of this as the internal abdominal ring, but as I have never seen an internal abdominal ring I prefer to call it the internal opening.

Next, the external opening is formed by the external abdominal ring, which, being made of aponeurotic structures attached to bony points, may dilate but cannot move. But now observe what happens to the internal opening in a neglected case. The contents of the hernial sac are most likely to consist of intestine or of omentum, or of both intestines and omentum. Now consider the attachments of either the mesentery or of the great omentum. Roughly speaking, these are in the middle line of the body. Also the external opening of the rupture is situated near the middle line of the body. Now observe the course taken by the gut and its mesentery or by the omentum. If the rupture is on the right side of the body they pass from the middle line of the body towards the right, enter the internal aperture situated just inside the centre of Poupart's ligament, descend the oblique inguinal canal, and then turn downwards into the scrotum. Pulling in the line of gravity and towards the middle line of the body, the contents of the hernial sac must tend to pull the internal opening towards the middle line. For a time this displacement is resisted by the posterior wall of the inguinal canal, but that is not very thick nor strong, so that in course of time the internal opening is pulled inwards until it corresponds with the external opening. Now a finger thrust into the external abdominal ring meets with no resistance and passes straight into the abdomen, and when it has turned inwards the outer end of the rectus abdominis is felt, the back of the canal and the conjoined tendon having been pulled against it. These changes in the inguinal canal have a direct bearing upon the question of operation and upon that of treatment by trusses.

I have several times referred to the contents of hernial sacs, but please do not think that I pretend to know what is inside a hernial sac. Years ago the great Petit warned us of the foolishness of attempting this impossible feat. I have in my hand the fifth edition of a treatise on ruptures by William Lawrence, who retired from the surgeoncy of St. Bartholomew's Hospital at the age of eighty-

four. Sir George Humphry told me that he considered Lawrence to have had the best intellect of any medical man that he had ever known—high praise from one of the most sagacious of human beings. Lawrence says that "Petit, after stating with a candour characteristic of true science that he had been frequently mistaken in his opinion, delivers the following very sensible observations which it may be well for the young practitioner to bear in mind on other occasions as well as the present. Let young surgeons acquire a habit of caution from what I now say, let them reflect before they speak or act, and remember that there is often a great difference between what a person really sees and what he fancies that he sees. Speaking too hastily may be followed by bitter regret, but we very seldom repent of having been silent. Those who run after reputation do not always overtake it. The merit on which it is founded is like fruit which ought not to be gathered until it has attained maturity." I remember once hearing a surgeon explaining to the students who were looking on that he was about to find an ovary in an inguinal rupture of the left side. I waited to see what supervened, and was rather amused when the sac was opened to see a lump of inflamed and indurated omentum slip out. That was the hernia of the ovary.

But to return after this digression to the condition of the inguinal canal, I am not concerned to-day with the question of radical cure of hernia, but clearly when the inguinal canal has retained its proper length and has a back to it the conditions are favourable for a radical cure. But when the inguinal canal has ceased to exist and the internal opening has been pulled opposite the external, it may be difficult or impossible to restore it to its former condition and make it what it ought to be —an oblique canal with sphincter action.

Now let us see how the condition of the inguinal canal bears upon the treatment by means of a truss. I place before you the type of truss which I advise you to thoroughly understand and use in your practice. It consists of a steel spring to encircle the pelvis and of a pad to press upon the inguinal canal. The spring is padded and covered with wash-leather. Wash-leather is comfortable to wear and does not slip upon the skin. The small oval pad is also covered with wash-leather and I have cut this one open to show you that it consists of a thick layer of felt. I am informed by Messrs. Krohne and Sesemann, who have kindly lent these trusses, that they use the best piano felt. Behind the felt and next to the expanded plate of the truss you will see that there is a further pad of cork. I do not think it is worth your while to devote your time to trying to learn other types of truss. I have never met with any so reliable as the one I show you nor do I advise you to prescribe trusses which have pads filled with air, water, or glycerine, for resistance is required to check the impulse of a rupture. In addition to the parts I have mentioned the truss is supplied with a perinæal band which goes round the thigh to prevent the pad slipping upwards on the abdomen.

Now let me show you how I should measure this young man for an inguinal truss, and how I should determine what kind of pad to give him. First, observe that he is thin. Thin people are not so easy to fit as those who have plenty of subcutaneous fat. A truss sinks into the fat and keeps its position. Observe, again, that the patient is lying down. If he were to stand up his pelvic measurement would be at least an inch more and the truss would not fit closely to his pelvis. Next I wish to draw your attention to the sharp angle which he has beneath the anterior superior spine of the ilium. The spring of his truss will have to be sharply bent at this point, otherwise it will not fit. Unless you understand the importance of the angle you cannot write a proper prescription for a truss. We have already determined that his inguinal canal is a good one—that is to say, that the internal opening is in its proper position. Now I pass the tape round his pelvis. Behind, it crosses the base of the sacrum. At the angles the tape is in the groove below the anterior superior spines of the ilium; it meets in the middle line at the symphysis pubis. I read off the measurement: it is thirty-four inches. Now we will write a letter to the instrument maker prescribing the truss, but before prescribing I have ascertained that the patient does not suffer from a cough, and that his work is not very arduous and does not entail heavy lifting. Thus he would not require a very strong spring to his truss. The letter ordering the truss would run as follows: "Kindly supply Mr. X— with a thirty-four inch inguinal truss, medium strength of spring and sharp angle, wash-leather cover. Kindly say when I can see this truss fitted on." When this truss is finished it is neces-

sary to teach the patient how to put it on and to see that it is a proper fit. It should always be put on the patient lying down. Teach the patient to put it round the thin part of his thighs, and after reducing his rupture pull the truss up to the pelvis and adjust the pad of the truss on the inguinal canal. The lower edge of the pad should be just above the crest of the pubes. The thigh strap should be adjusted rather loosely. Whilst the patient is lying down the truss may look too big, but as soon as he stands up the abdominal wall falls forward and fills the hollow. Now ask the patient to cough. The pad of the truss pressing the exterior and posterior walls of the inguinal canal together will prevent the descent of the rupture. Now request him to sit down and cross his legs and see whether this disturbs the pad. Then request him to quickly stand up. As a rule, it is not necessary for the patient to wear the truss at night unless he has a cough. But when you are quite sure that the truss fits and is comfortable it is wise to obtain another as a stand-by. A well-made truss lasts from a year to eighteen months, after which it may want re-padding or the spring may be worn out.

If I may be allowed to criticise these excellent trusses made by Messrs. Krohne and Sesemann, I should say that it was better that the pads should not be fastened to the spring by means of screws. Patients are very odd. It seems impossible for some to refrain from using screw-drivers and altering the pads which you have carefully adjusted.

As you are aware, it is useless and most improper to wear a truss which does not keep the rupture up. Nevertheless, after pointing out to patients that their truss was useless, and probably doing harm by pressing upon the unreduced rupture, they have carefully put it on again on top of the unreduced rupture before leaving my consulting room.

I now show you what the instrument makers call a scrotal truss. It has the same spring as the inguinal truss which I have just shown you. But the pad, you observe, is large and triangular. Supposing as the result of your examination you had found that the internal and external openings of the inguinal canal had come together so that the finger passed straight into the abdomen, then it would be useless to order an inguinal truss. Under these circumstances it is much more difficult to retain a rupture by any type of truss. But as a rule you will succeed with a scrotal truss—that is to say, with one of those trusses with a triangular pad. So that in ordering a truss for a person with a direct opening you would write a prescription in the same way, but you would say—"Kindly supply Mr. X— with a right or left or double scrotal truss." Then you would give instructions about the spring, the angle, and the covering. The object of a scrotal truss is not to press together the anterior and posterior walls of the inguinal canal because the posterior wall does not exist, but it is to occlude the opening through which the rupture descends. The truss is put on the same as inguinal trusses, but the pad is adjusted over the opening and near the middle line of the body. If when the patient coughs the rupture still descends, note exactly where the protrusion takes place. Sometimes the protrusion is below. In that case, by bending the plate of the truss backwards this may be prevented. At other times the protrusion is at the inner side. To stop this, move the pad farther towards the middle line—and here one of the advantages of the movable pad becomes apparent. If this does not succeed I have seldom failed by having the inner edge of the pad heaped up so that it almost forms a ridge. Sometimes the patient cannot wear a truss because it gives rise to pain. This may be due to the presence of a slip of adherent omentum within the sac. There is no way of detecting this peculiarity except by opening the sac. But you may suspect it when the patient informs you that although he has tried five or six or more types of trusses, he has been unable to wear any of them. Sometimes an inguinal truss cannot be worn because of the presence of an inguinal varicocele, a subject upon which I have already lectured. The other day a patient could not wear a truss because the sac was inflamed and thickened. At other times a truss cannot be worn because there are lumps of subperitoneal fat about the neck of the sac. At times you will find a patient will be much more comfortable if you prescribe a double inguinal or double scrotal truss, although he may be ruptured on one side only. A double truss has rather a longer spring and the pressure seems to be better distributed. Also, it is easier for the patient to adjust. However, when you order a double truss it is well to tell the patient that he is not to infer that, therefore, he has an unusually bad type of rupture, or that because you have ordered a double truss you think he has a double rupture. I do not exactly know what a double rupture is. I suppose that to medical men it means that the patient is ruptured on both sides. But I am sure that to others it has some other occult meaning, and implies a horrible state of affairs.

June 27th, 1910.

SYPHILIS.

A Discussion * by

Mr. Ernest Lane, Mr. J. E. R. McDonagh,
Mr. Alexander Fleming, Mr. C. F. Marshall,
Major H. C. French, R.A.M.C., Dr. Pernet,
Mr. H. W. Bayly.

Mr. Ernest Lane: As far as our present knowledge is concerned, syphilis is a malady which can only be mitigated, and which does not seem likely ever to be exterminated. Its ravages at the present time are still sufficiently in evidence as to justify its being considered a subject worthy of discussion by the Surgical Section of the Royal Society of Medicine, though in contrast with a quarter of a century ago they are comparatively trifling, in that the disease is seldom now productive of the terrible mutilations with which we were familiar in the past. Still, though the more grave external and palpable evidences of late syphilis are not so frequently encountered, yet its remote effects, its influence on the nervous system and on many medical and surgical conditions cannot be ignored. It is a matter of the utmost difficulty to gauge the incidence of a disease such as syphilis, and a glance at hospital records might give the impression that it was on the increase; for example, the attendances of out-patients at the London Lock Hospital was in 1908, 34,649, whereas ten years previously the number was 25,314, a difference of over 9000 attendances. These figures, of course, only apply to venereal diseases in general, and not to syphilis in particular, but the ratio of syphilis to other venereal diseases appears to remain fairly constant. This increase in the number of cases treated does not necessarily mean an increase in the amount of syphilis, but may be ascribed to the fact that the public nowadays more fully recognises its gravity, and the necessity for prolonged treatment. More reliance may be placed on the returns issued by the Army Medical Department, which show a steady decrease in the amount of venereal disease in the Army in the course of the last quarter of a century.

Taking it for granted that the extent of syphilis is considerably less than formerly, and that it is of a less virulent type, one is led to inquire into the causes for this diminution, both in its amount and in its gravity, and the conclusion that will probably be arrived at is that it is due partly to a more efficient system of treatment, partly to the better recognition by the general public of the laws of hygiene, of the dangers of the disease and of the disasters that may occur if treatment is neglected, and partly due to the process of natural immunity which after the lapse of years must show its influence on any disease. One of the best measures of prophylaxis against syphilis is efficient treatment, and it is to this point that my humble contribution to this discussion will be devoted.

There was until recently a considerable difference of opinion as to the exact period when the treatment of syphilis ought to be commenced. Some counselled the administration of anti-syphilitic remedies as soon as the diagnosis of syphilis was arrived at; others, recognising that it was often a matter of the greatest difficulty to make a diagnosis of syphilis from the appearance and feel of the sore, postponed the commencement of treatment until the appearance of some secondary lesion rendered the diagnosis a certainty. There is no doubt that in the past, through errors of diagnosis, many patients who had never suffered from syphilis have been submitted to treatment for that disease, protracted according to the methods of the individual surgeon. But at the present time such a mistake should be impossible, owing to the momentous discovery of the *Spirochæte pallida* by Schaudinn in 1905, and by the subsequent investigations by Wassermann on the serum diagnosis of syphilis. If the chancre exists, the presence or absence of the spirochæte will prove its nature, while if the sore has healed, then the Wassermann test will clear up any doubts.

It is undoubtedly to the material advantage of the patient that treatment should be commenced at the soonest possible opportunity after a positive and not a putative diagnosis of syphilis has been arrived at, and an early recognition of the spirochæte in the discharge from a venereal ulcer is a conclusive proof as to the nature of the disease. I have seen a sore of only one day's duration, or possibly recognition, in a patient who had exposed himself to contagion, and who had been keeping a keen look-out for possible contingencies; this sore was of the minutest dimensions, and might have been a simple abrasion, might have been a commencing chancroid, or might have have been her-

* Before the Surgical Section of the Royal Society of Medicine.

petic in nature, but on microscopical examination the spirochæte were found in large numbers in the exudation from the sore, and consequently I was enabled to place that patient on anti-syphilitic treatment almost immediately after the appearance of the initial lesion, and long before a diagnosis could have been arrived at without the discovery of the syphilitic organism.

The diagnosis having been reduced to a certainty by the microscope or by the serum test, the next question is as to the most efficient method of treatment to be pursued. The treatment of syphilis by mercury dates back more than five hundred years, and though that drug has occasionally been threatened by therapeutic rivals, it still maintains its position of supremacy, from which it does not at present seem likely ever to be displaced. But though it is universally recognised as the most powerful anti-syphilitic remedy at our disposal, yet there is great diversity of opinion as to the methods by which it should be administered. Personally I prefer not to limit myself to any one particular method of treatment, and do not consider that one routine plan is applicable to every case of syphilis, but I would prefer to treat every case on its merits, and according to individual peculiarities. Fortunately, in the majority of cases syphilis is a benign disease, and one which is particularly amenable to treatment, and in a simple case there is no necessity to look beyond the time-honoured method of administration of mercury by the mouth in the form of pills or mixture, and most cases can be conducted to a satisfactory conclusion by this, the most simple and most convenient of plans.

But it is not every case of syphilis that will yield to such simple measures, and it is not every patient who is tolerant of mercurial pills or mixtures. One is often called upon to treat some severe manifestation of syphilis which may be threatening the integrity of some important organ, such as the eye, which may be causing progressive mutilation and disfigurement, or which may be involving the nervous system or other regions of equally vital importance. Again, one may meet with cases in which the digestive organs are intolerant of, and rebellious against, any form of mercury administered by the mouth. For such as these there are other means at our disposal, which may by their potency contend with the immediate emergency, and may not in any way interfere with the digestive functions. First and foremost may be mentioned the method of intra-muscular injections of mercurial preparations. I need not weary you by enumerating or discussing the relative merits of the legion of preparations which have been recommended for intra-muscular injections, but will proceed to the consideration of this plan of treatment. Intra-muscular injections may be given in the form of the insoluble or the soluble preparations of mercury, and of the two the insoluble salts are undoubtedly the more valuable, for they produce results which are surprisingly alike in the rapidity with which they bring under control manifestations which had previously resisted other kinds of treatment, as in controlling the subsequent course of the disease.

The two reliable preparations of the insoluble salts are, first, calomel, and secondly, mercury suspended in some fatty basis and usually designated grey oil, though occasionally christened by the name of someone who has devised some modification of the compound originally introduced by Lang, of Vienna. Of these two methods, that of calomel injections is far the more efficient, indeed, they constitute by far the most powerful anti-syphilitic remedy at our command. The dosage of the calomel injections will vary according to the condition, the weight, the age, and the sex of the patient, but the quantity usually administered at each injection is gr. $\frac{2}{3}$, suspended in sterilised olive oil, or some similar vehicle. A course of twelve injections given once a week is usually sufficient to clear up any urgent symptoms, and to check the progress of any rapidly destructive lesion. In appropriate cases the effect of intra-muscular injections is most remarkable, and this plan of mercurial treatment is usually successful when all other measures have failed to check the progress of the disease. Amongst the conditions to which this treatment is applicable are cases of malignant syphilis attended by severe ulceration and entailing disfigurement and mutilation; syphilitic iritis, threatening impairment or destruction of vision; cerebral or spinal syphilis; tertiary syphilitic laryngitis, and pharyngitis; chronic and inveterate syphilides, resisting the ordinary methods of treatment; chronic glossitis, and that terribly disfiguring condition, syphilitic macrocheilia. For the past four years I have employed calomel injections systematically in my hospital practice, for the reasons that the disease is more rapidly got under control,

that the patient's stay in hospital is thereby rendered shorter, and that the subsequent course of the disease is distinctly modified by this early and energetic attack upon the poison. The objections which have been advanced against this method of treatment, in addition to those which are inseparable from every plan of introducing mercury into the system, such as stomatitis and gastro-intestinal complications, are severe pain, possibly abscess, pulmonary embolism, and sudden and acute mercurialism. It cannot be denied that the pain following upon these injections is occasionally very acute, and it may be prolonged for several days, but this is by no means an invariable occurrence, and in a certain proportion of cases it is a negligable quantity; for instance, I have come across several patients who, immediately after having received their injection, have proceeded to play a round of golf. As regards abscess, I have met with several cases of severely painful and fluctuating nodosities of the buttocks supervening shortly after the introduction of the injection, but with one exception these have been relieved by aspiration and by the withdrawal of a collection of chocolate-coloured fluid, and the subsequent introduction of some boracic lotion. Cases of pulmonary embolism I have not so far encountered, though many fatal instances of this disaster have been recorded in the past; they were due to the direct introduction of these insoluble compounds into the gluteal veins or their tributaries, and must be attributed to neglect of the anatomical position of the gluteal vessels. There is a zone of safety for intra-muscular injections into the buttock, and unless this safety zone is departed from, I cannot conceive the occurrence of this severe and alarming complication.

By the use of calomel injections a reservoir of mercury is introduced into the system, into which it should be absorbed gradually, but it occasionally happens that these injections become surrounded by an inflammatory zone, which for some reason or other may suddenly give way, with the result that a large quantity of calomel may suddenly be released into the circulation, and symptoms of acute mercurialism may supervene.

Another insoluble mercurial preparation which has achieved much popularity is grey oil, consisting of mercury very finely subdivided and combined with some fatty basis. Its action resembles that of calomel, but it is not nearly so powerful and falls far short of it in efficiency, though it has some compensatory advantage in that the resulting pain is much less severe. Whilst calomel may be employed in the intractable cases, in those in which some serious complications are present, grey oil is more suitable as a routine treatment, and for this reason is more in vogue in Military and Naval hospitals, where the patients can be kept under constant observation and supervision.

I shall not devote much time to the treatment by injection of soluble mercurial preparations, for its drawbacks are so obvious and its advantages so visionary that the method compares unfavourably with all the other means by which mercury may be introduced into the system. The principle objection to this method is the number requisite to constitute an efficient mercurial course, for the treatment involves a series of from thirty to forty injections administered daily, and consequently few patients will be found willing to submit to it on the grounds of inconvenience and expense; further, there is the constantly recurring pain after each injection, not so severe, however, as after the use of the insoluble compounds. Another familiar procedure for the introduction of mercury into the system is that by inunction, a very effectual means of getting a patient rapidly under the influence of the drug, but one which, in my opinion, does not compare for efficiency with intramuscular injections of the insoluble mercurial salts, and which has certain counterbalancing disadvantages, which have, in this country at any rate, relegated it to a position of comparative obscurity. Its action is prompt, and it leaves the stomach at our disposal for the ingestion of other anti-syphilitic remedies should they be called for; but the same applies to intra-muscular injections, and it cannot be denied that the suggestion of inunctions is repugnant to the feelings of a large number of patients. The principal objections to the inunction treatment are, that the patient is compelled to regulate his habits according to the requirements of the treatment, and has to follow out a *régime*, which must perforce somewhat interfere with his daily avocations. The necessity of retiring to rest with a moiety of the body besmeared with ointment is an unsurpassable objection to a sensitive and æsthetic patient. If the skin is very delicate inunctions may give rise to a troublesome

eczema, while if it is more than usually hairy, localised suppuration in the hair-follicles may ensue; further, in the presence of extensive cutaneous manifestations of syphilis it is inadmissible. There is also great uncertainty as to the amount of the drug which will be absorbed, and in some cases its action appears to be absolutely inert. Finally, it will not commend itself to the large number of patients who are anxious to conceal the nature of their disease from their friends and relations.

Other methods of administration of mercury are at our disposal, such as fumigations, rectal administration, intravenous injections, and the inhalation and consequent absorption of mercury by the wearing next the skin of flannel or cotton-wool impregnated with the drug in a state of fine subdivision.

One of the most important and vital questions is the length of time during which anti-syphilitic treatment should be prolonged; the general impression in this country appears to be that mercurial treatment prolonged for two years is sufficient to guarantee, if not a cure, a subsequent immunity from symptoms. Such a supposition is, in my opinion, fallacious and dangerous, as is evidenced by the large number of cases of late syphilis, specially involving the higher nerve centres, with which anyone who is in the habit of treating the disease must be painfully familiar. The ultimate object of every method of treatment which may be employed is to render the patient impregnable to the ravages of late syphilis, to prevent the occurrence of tertiary symptoms, and especially to guard him from the disastrous effects of syphilis involving the cerebro-spinal centres, or important internal organs, such as the heart, the lungs, the kidneys, and the liver. We often dismiss a patient as cured, and consider that we have been instrumental in eradicating the baneful poison from his system; not hearing of any recurrences, we conclude that we have effected his cure. But only too often symptoms crop up in later years, which the patient may not in any way associate with his more youthful indiscretions, and for which he may consult the neurologist, the ophthalmic surgeon, or those eminent in some particular branch of their profession. The late or tertiary manifestations of the disease are constantly coming before our notice, both in hospital and in private practice, and they are the result either of the insufficient duration or of the neglect of the treatment by the patient himself. I have for a long time past recommended that treatment should be prolonged over a minimum period of five years, and in the case of those who are of a neurotic disposition, and whose business entails a strenuous and anxious life, I advocate that it should be extended over seven years. Obviously the treatment cannot be continued for all this length of time, but should consist of one initial course, extending, if possible, over a year, then an intermission of three or four months, and then a series of shorter courses of treatment and more lengthy periods of intermission until the expiration of the number of years deemed necessary for each individual case. Long before the expiration of that time the patient will in most instances have been incapable of transmitting the disease to his wife or to his offspring, but though he may have attained this point, he is not himself necessarily immune from the occurrence of nerve or visceral syphilis. We are not yet sufficiently conversant with the exact significance of the Wassermann test for syphilis; no doubt the test often remains positive long after the patient has ceased to be a source of danger to others, but if positive it appears to me to be a sufficient indication that the patient will be benefited by further courses of treatment until the test became permanently negative.

I shall not dwell at any length on treatment by the iodides, which are of the greatest value in getting rid of certain syphilitic manifestations, though they do not appear to be capable of eliminating the poison of syphilis in the slightest degree. Some patients appear to be unusually susceptible to the action of iodides, and show symptoms of iodism very shortly after the treatment has been commenced. Many modifications of iodine have been suggested by which the disadvantages and discomforts of iodism could be obviated or modified. One preparation which has attracted favourable attention is iodipin, a compound of iodine and sesame oil; this is said to be more efficacious in the form of intra-muscular injections than when administered by the mouth, as more iodine is liberated into the system by the former method. This preparation is hardly so popular here as at continental health resorts, such as Aachen, where it is given in enormous doses,

such as 3x (ten drachms) of the 25 per cent. solution daily, until as great a quantity as forty ounces has been introduced in the space of two months. I have seen cases in which the administration of this substance was productive of excellent results; but on the other hand I have seen a patient who suffered from almost complete atrophy of the gluteal muscles in consequence, and another who was leaking out iodipin from many punctures in his buttocks, to *his* great embarrassment and disgust, and possibly to that of others.

For many years past arsenic has been recognised as a valuable adjunct to mercurial treatment, especially in the form of Donovan's solution, a preparation which is specially indicated in the treatment of late and intractable syphilides. But the amount of arsenic that can be introduced into the system by way of the stomach is very limited, the dose being from gr. $\frac{1}{60}$ to gr. $\frac{1}{15}$, and any larger doses are liable to be followed by signs of arsenical poisoning. In order to facilitate the introduction of larger quantities of arsenic into the system, certain compounds known as the arylarsonates have of late years been introduced, and have met some amount of success and support in the treatment of syphilis. I need not trouble you with details as to the composition of the various arylarsonates, but will enumerate those with which we are most familiar in this country. They are atoxyl, containing 27·3 of arsenic; soamin, with a percentage of 22·8; arsacetin, 21·25 per cent., and orsudan, 25·4 per cent., to which may be added another preparation known as atoxylate of mercury, containing 23 per cent. of arsenic and 31·8 per cent. of mercury. Of these preparations, the two with which I am most familiar are soamin and orsudan, both of them products of a firm of the highest repute, and I have notes of forty-four cases treated by the latter preparation. The dose I used was ten intra-muscular injections of orsudan gr. x introduced on alternate days, so that by the end of nineteen days over twenty-five grains of arsenic had been introduced into the system. The success obtained by this treatment was at first most encouraging, and certainly it caused the disappearance of the early symptoms of the disease with wonderful rapidity. In very few of the cases were symptoms of arsenical intolerance observed, and these disappeared on prolonging the interval between each injection. But eventually I met with a catastrophe so grave, and so disturbing, that since that event I have entirely discarded this form of treatment. A patient, æt. 49 years, an old soldier, was admitted to the Lock Hospital suffering from a typical indurated sore and a papular syphilide on the face and trunk. He was given nine injections of orsudan gr. x on alternate days; on the day after the seventh injection he noticed general fogginess of vision in both eyes, which gradually increased, but he omitted to mention this fact until the day after the ninth injection. Then on examination there was found to be tremor of both eyes, and difficulty in counting fingers held up at a distance of twelve inches in a strong light. On ophthalmoscopic examination both discs were found to be pale and indistinct in outline, with the vessels of normal calibre. A very careful general examination showed absence of any signs of arterial degeneration, the kidneys were apparently perfectly sound, and the physique and general health were perfectly good. He was treated with large doses of strychnine hypodermically, and subsequently with calomel injections. Examination of the eyes four months later showed a perception of hand-movements at a distance of four feet, equal in both eyes; both discs were markedly atrophic, the atrophy not indicative of a previous neuritis, whilst there were well-marked pigmentary changes at both maculæ suggestive of inflammatory or degenerative changes. Shortly afterward I met with another very similar case of a patient, æt. 46 years, who had been treated by a private practitioner with injections of soamin; he had received ten injections of soamin gr. v on alternate days; after the third injection he noticed some haziness in vision, more marked in the right eye. After the sixth injection the impairment of vision became more marked, but he could still see objects at a distance, though definition was not good. After the first course of soamin all signs of syphilis had disappeared, and treatment was stopped for two months. He then was given five further injections this time of gr. x of soamin on alternate days, but then the vision became so obscured that he became alarmed, and declined any further injections. Assuming that his ocular symptoms were attributable to syphilis he applied for and obtained admission to the Lock Hospital. He then had no signs of syphilis, his general health was good, there was no sign of arterial disease, and both skin

and kidneys were satisfactorily performing their functions. On examination of the eyes there was found to be perception of hand-movements at six inches in the right eye and eighteen in the left; both discs showed typical primary optic atrophy, and there was no alteration in the retinal vessels, and no alterations in the maculæ. I felt it my duty to draw the attention of the profession to the danger which might ensue from the employment of these preparations, and communicated with one of the medical journals on the subject. My letter was followed by various comments, some unfavourable, and accusing me of creating an unnecessary panic, others agreeing with me that my warnings were perfectly justified. In confirmation of my warning, I learn that the treatment has been discontinued by such authorities as Professors Neisser and Finger, owing to the occurrence of similar mishaps. It cannot be denied that the results of this treatment have been satisfactory in some of our military hospitals, but opinions as to the value of the arylarsonates differ widely amongst the officers of the R.A.M.C. Whereas some regard it as a prophylactic against any further developments of the disease, a contrary opinion is expressed by a memorandum issued in January of this year by the expert committee on venereal disease in the Army to this effect: "That the utility of these preparations as a prophylactic against syphilis has not been proved, and the Committee does not recommend their administration for that purpose."

It seems to me to amount to this, that the arylarsonates do in a certain number of cases considerably modify the evolution of the early symptoms of syphilis, but they are just as liable to be followed by relapses as is the treatment by mercury, which also appears to be more reliable; that we have in the various methods of administration of mercury a perfectly satisfactory remedy for the treatment of the disease, and one which, with ordinary care, is at any rate free from danger. In the presence of a disease such as syphilis, which in the vast majority of cases is amenable to mercurial treatment, the effects of which, though slow in many cases, are for the most part sure and safe, I cannot think we are justified in having recourse to remedies such as these arsenical preparations, which are capable of causing such catastrophes as those I have already alluded to. But with a disease such as trypanosomiasis, which is almost certainly fatal unless drastic measures are resorted to, the employment of these remedies is not only justified but is imperatively called for, since the possibility of blindness is better than the certainty of death.

Mr. Alexander Fleming: My contribution to this discussion will not deal directly with the treatment of syphilis, but will be directed to show in what ways laboratory work can aid in such treatment. First, we must deal with the diagnosis of syphilis, for of course it is only by means of accurate diagnosis that we can conduct successful treatment. In most cases clinical observation is all that is required, but there are always some cases where the clinical signs are not definite, and where, if treatment is to be started early, other tests are necessary.

The laboratory diagnosis of syphilis can be divided into two parts: (1) Finding the organism —the *Spirochætu pallida*; (2) performing the serum test—Wassermann's reaction. The organism can rarely be discovered except in the primary and in some secondary lesions, as in the other cases either the lesions are in parts of the body that are not accessible, or the organisms are so few and far between that they cannot readily be found. The methods which have been used to demonstrate them are these: (1) Staining methods; (2) cutting sections of a piece of material removed by operation after impregnation with silver; (3) dark-ground condenser; (4) Chinese ink (Burri's method). The first two I will say no more about, as they have been almost entirely replaced by the other methods. The dark-ground condenser has come largely into use for this purpose in the last year or two. By this means we are able to see the spirochætes as very definite clear spirals on an absolutely dark ground. This method also has the advantage over all the other methods that one is able to see the characteristic movements of the organisms. The disadvantage of the method is the necessity of obtaining a special condenser, and of fitting it into one's microscope each time one wants to look for the spirochætes.

A much easier method and one which requires no elaborate materials and very little skill is Burri's Chinese ink method. Here all one has to do is to put a small drop of Chinese ink on a slide, mix with it some serum squeezed from a chancre, and

spread the whole on the slide in a thin film. The film is allowed to dry and is examined directly under the microscope. I have found that this method enables me to find the spirochæte with as much certainty and with much less trouble than by the use of the dark-ground condenser. The dis advantage is that it does not reveal the movements of the spirochæte.

The second test for syphilis is the serum reaction. While the spirochætes are most readily found in the primary stage, it is in this stage that the serum reaction is of the least value, although even here it gives positive reactions in between 60 and 70 per cent. of cases. In the secondary stage, where lesions are present, one obtains a positive reaction in almost every case, and the same may be said of the tertiary stage where there are any active lesions. Out of about a hundred cases of secondary syphilis, I have only found one, or possibly two cases, where I did not obtain a definite positive reaction. In congenital cases also I have found that out of about forty cases I have only obtained one negative result where the clinical diagnosis seemed quite definite. In parasyphilitic cases there is a very marked difference between general paralyis and tabes in respect of the serum reaction, for whereas in general paralysis one obtains a positive reaction in every case, in tabes it is in only about 65 to 70 per cent. that that reaction is shown.

In making my observations on the serum reac tion I have used, not Wassermann's method, but a method which I demonstrated before the Clinical Section of this Society in May of last year. This method is in principle the same as that of Hecht, although the technique is different, and as certain objections have been raised to the method of Hecht these objections also apply to this method, and I will try to answer them. The most serious objection which has been urged (mainly on theoretical grounds) is that by this method one will obtain a positive result in some cases which are not syphilitic. If this were so, then it would render the method almost valueless, but I strongly maintain that this objection is absolutely devoid of foundation. I have now tested over 500 individuals who were supposed to be free from syphilis. Some of these were patients attending the hospital for diseases other than syphilis, and others were workers in the laboratory, and visitors. Of these 500 cases, only some twelve gave positive reactions, and on going into the history of all of these, facts were elicited which made it very probable that in all there had been a syphilitic infection. In no other disease with the exception of leprosy and trypanosomiasis have I found a positive reaction. I think it is likely that the observers who found positive results in normal people were either using a faulty antigen or were using it too strong, as of course, by using too strong an antigen, one could easily make all of us give a positive reaction. Last July, at the British Medical Association meeting at Belfast, Fleet-Surgeon Bassett Smith made some objections to the method I have used, but later, in the 'British Medical Journal,' he stated that as the result of further experience his objections had been unfounded, and that by this method he had obtained in some cases a positive result earlier in the disease than he had obtained by the original Wassermann method. The most recent figures comparing the two methods are those of Hecht in the 'Zeitschrift fur Immunitats-forschung' last month, which I quote.

	Both methods +.	Both methods −.	Wasser-mann +, Hecht −.	Wasser-mann −, Hecht +.
Syphilis (primary, secondary, tertiary, and congenital)	218	42	1	23
Syphilis (latent) . . .	33	27	—	16
Non-syphilitic . . .	1	141	—	—

From these it will be seen that not only is the simpler reaction absolutely specific but that it is a more delicate reaction, for whereas in only one case did Wassermann's method indicate syphilis where Hecht's method failed, in 39 cases did Wasser mann's method fail where Hecht's method gave a true indication of the disease.

But apart from any question of greater accuracy of the modification I have used there can be no question as to its greater practicability. All one requires is some extract of heart, some serum (the amount which can be obtained in an ordinary blood-capsule) and some sheep's corpuscles, which can be obtained from the butcher, whereas in Wassermann's method one requires in addition some serum of a rabbit which has been immunised to sheep's corpuscles and some fresh guinea-pig's

serum—serious drawbacks if one does not possess a vivisection licence. The place of the rabbit and the guinea-pig are taken by the hæmolytic substances which are present naturally in human serum, and in the few cases where this hæmolytic power is deficient (about 5 per cent.) it can be supplied by the addition of a little fresh serum from a normal individual. In addition one does not require to heat the serum for half an hour to 56°, as is necessary in Wassermann's method.

Mr. McDonagh, in a recent paper in the 'Lancet,' emphasises the fact that one of the great reasons why Wassermann's reaction did not succeed in all cases of florid secondary syphilis, was that in heating the serum previous to the test the complement of the serum was converted into complemoid which possessed the combining properties but not the active properties of complement. If this is so, then it puts Wassermann's method at a very great disadvantage to Hecht's, in which the serum is not heated.

Possibly the greatest use of the serum reaction will be to control treatment. There seems no doubt that energetic treatment with mercury will destroy the reaction. If treatment is stopped too early the reaction will return after an interval of a few months, but if treatment has been energetic enough, or if it has been sufficiently prolonged, then in the majority of cases the reaction seems to have disappeared for good. Of course, owing to the short time that the reaction has been in use, we have not had an opportunity of following cases for a sufficiently long time after treatment to make this absolutely certain, but this is the impression that we get from testing cases at varying lengths of time after treatment has ceased.

The action of mercury on the spirochæte is possibly like what Ehrlich found in connection with atoxyl and trypanosomes. Energetic treatment with atoxyl quickly killed most of the trypanosomes, and further treatment with the same drug was of no avail, as the remaining trypanosomes seemed to be in some way protected against the atoxyl, so that it was only by the substitution of other drugs that they could be in any way affected.

For some months past I have been making observations on the effect of treatment on the serum reaction, and as the cases came from different sources I have been able to make some comparison between the different forms of treatment employed. The number of cases is, however, comparatively small, and further observations may quite alter my conclusions.

It appears to me that the most effective treatment is by injection of grey oil, as with this form of treatment a fair number—about 30 per cent.—lose their reaction after only nine months' treatment. I have, however, not tested any cases treated for more than twelve months in this way, and possibly, although the reaction seems in some cases to disappear fairly early, yet in the long run no greater percentage will lose it than with other forms of treatment. After grey oil injections, inunction of mercury seems to have the most effect.

Treatment by means of pills of hyd. c. cret. appears to be the least effective of the methods I have made observations on, as even after three years' treatment in this way more than half of the cases tested still gave positive results.

Lastly, as regards the procedure to be adopted in syphilis, I would put forward that after the surgeon has treated the case for a reasonable time, he should stop treatment for a month or two and then have the serum test done. If this is positive then treatment should be recommenced; if negative, the test should be repeated after an interval of six months or a year, and if the patient wants to be further reassured, the test may be repeated at later intervals.

Major FRENCH: Our position in regard to the treatment of syphilis is, I am afraid, too often influenced, unless the cases are in hospital, by the convenience of this or that individual. To adequately treat syphilis, and so keep the disease under effectual control, it appears to be absolutely essential to recognise it in the earlier and more remediable stages. If the *Spirochæta pallida* is demonstrated, or if the classical sign of induration exists in the chancre and in the adjacent lymphatic glands, we should, I consider, vigorously attack the disease by *judicious* mercurial treatment. The Wassermann serum test cannot be implicity relied on in the primary stage. I do not agree with those persons who counsel a waiting policy in all cases until the rash is present. The rash, in my experience, does not as a rule appear until fourteen to sixteen weeks form the date of exposure to contagion. Delay for this period in

commencing treatment is unjustifiable in probably 90 per cent. of cases, and markedly aggravates the disease. In a small minority, however, where induration of the chancre and discretely enlarged lymphatic glands are not marked, we must perforce wait for more definite evidence, such as rash, etc.

A course of mercurial treatment before the date of onset of rash is, I consider, worth two courses later as regards the ultimate beneficial effect on the disease, as well as in its effect in guarding against parasyphilitic affections. As regards *duration* of treatment, a period of two years with intervals between courses is my usual rule—where inunctions or hydrarg. cum creta with opium have been used—but when insoluble preparations of mercury, such as grey oil, are employed, a third year's course of treatment is often necessary to dissipate the remaining external evidences.

Relapse, or accentuation of manifestations, whilst actually under treatment is, in my experience, rare with the inunction or hydrarg. cum creta with opium methods in hospital, but quite commonly observed under treatment by insoluble preparations of mercury. An increase of inefficiency in hospital consequently results, which is important in the Army from the point of view of the State.

Inunctions of unguentum hydrarg. form the routine method of treating syphilis in the French, German, Italian, Bavarian, Hungarian, Swedish, and Danish armies; also at Aix and Wiesbaden and Unna's and Pentippidan's clinics on the Continent, and Harrogate in this country. This valuable method is now receiving much more attention in the British Army. I employ it in the majority of cases in doses of two thirds to one drachm, with a daily hot bath (preferably sulphur), and thirty to fifty rubbings form a course in early syphilis, according to the indications of the particular case and controlled by the weekly weight. The patient must be seen daily by the medical man, and be kept in hospital. A mouth-wash of acetate of alum is used every two or three hours—and all carious teeth extracted or attended to carefully—and tooth powder used twice daily. All the minutiæ of technique must be rigidly adhered to in order to attain good results, and the patient well fed with milk and eggs in addition to ordinary food.

The rapid disappearance of induration in the chancre and glands is the best test of progress, and where this has occurred the disease can be well kept in hand by the avoidance of smoking, and local treatment if mucous patches develop, until the second course of treatment some six weeks later by inunction or other methods. In the treatment of mucous patches in the mouth, ulcers on the tonsils and laryngeal affections, the value of soft, non-irritating food, such as meat stews, etc., cannot be too strongly insisted on.

The Wassermann serum diagnosis test, according to the evidence of Captain Gibbon, R.A.M.C., recently working on cases in my clinic in Malta, is by no means always conclusive before the development of the constitutional signs of rash and generalised glandular enlargement. In several cases (where treatment was not employed) there was well-marked induration of the chancre and proximal lymphatic glands, the Wassermann test was negative, and syphilis supervened. In other instances, in which there was no clinical evidence of syphilis and no treatment employed, the Wassermann test was *positive* and syphilis did not later supervene. The technique employed was that recently outlined by Captain Gibbon in the 'Journal of the Royal Army Medical Corps,' where he gives 98 per cent. of *positive* results in cases with well-marked clinical evidence of the disease, the majority of which I had recently placed on the syphilis register.

The Wassermann test gave valuable results in parasyphilitic cases. The result was frequently positive after two to three years' mercurial treatment, and this is in consonance with the views recently expressed by Harrison, independently working at the Rochester Row Military Hospital, London, and quoted in the 'Journal of the Royal Army Medical Corps,' March, 1910. The inconclusive nature of the Wassermann serum diagnosis test in the *early* stages of syphilis before secondary (constitutional) manifestations of the disease have appeared is also recently outlined in the "Epitome" of the 'British Medical Journal,' and also on May 7th, 1910.

The foregoing and following facts were dealt with in detail in an article in the 'British Journal of Dermatology,' in November and December, 1908, and the 'Lancet' of September 24th, 1909, and January 8th, 1910, so I do not dwell at length

on them. The average manifestations in syphilitic cases are mild, and hence new remedies are very apt to be unduly exploited to the possible detriment of older, well-tried, and probably better methods.

The marked reduction of syphilis in the British Army in recent years has mainly occurred in India, not from the use of insoluble grey oil, but as the result of the control of diseased men and women under the excellent and moderate provisions of the Cantonment Code of October, 1897, as well as by systematised hospital and out-patient treatment. There has been a corresponding reduction in gonorrhœa and non-infecting chancres. I understand that in London there are less than 100 beds for the treatment of venereal disease in civil hospitals, whereas in Paris there are 2000. Consequently, disease is unduly and quite unnecessarily spread, and facilities in England for obtaining the "*connaissance approfondie*" so requisite for adequate treatment, and insisted on by the Brussels International Conference of 1899, are unduly curtailed.

Most persons will admit that iodide of potassium is a valuable drug in treating certain phases of syphilis, and its utility more obvious the longer the period from the primary evidence of the disease. I do not think it should be used to the exclusion of mercury, but to supplement it. My experience of soamin and arsacetin is that they exert a tonic action in the early phases of the disease, and commonly cause a temporary gain of five to seven pounds in weight amongst cases treated in hospital—but in many instances severe relapse has occurred with rapid loss of weight, and fits of an epileptiform character ensued.

The risk of blindness from optic nerve atrophy has been not infrequently recorded by other observers after the use of soamin and arsacetin in treating syphilis as well as with atoxyl in treating sleeping-sickness. The latter drug has caused complete blindness in fifty-four cases and partial blindness in one hundred and thirty-six cases (*vide* p. 1097, 'British Medical Journal,' May 7th, 1910).

In London hospitals I would suggest the provision of beds and the establishment of professorships as in the United States for this particular branch of medicine. In the Army, with the marked reduction that has occurred in the past ten years, venereal diseases even still cause 27–30 per cent. of the "constantly sick" in hospital from all diseases in all parts of the world. What is true of the Army would no doubt equally apply to the relative incidence in civil practice. A Royal Commission reported in 1889 that the cost to the State of maintaining and educating 7000 blind persons in the United Kingdom whose blindness resulted from a preventable disease, ophthalmia neonatorum (due to gonorrhœa), was £350,000 a year.

I would also suggest the appointment of a committee of medical men in England to collect definite views on all these various points on a somewhat similar basis to the valuable Advisory Board Reports on Venereal Diseases in the Army, published in 1905–6. Having collected these views, to memorialise Parliament to adopt suitable measures whereby this disease is not so largely spread in view of the immense cost to the country in maintaining lunatic asylums, hospitals, and syphilised persons in poor-house infirmaries and prisons. In some countries in Europe medical men, lawyers, clergymen, and others combine to form societies to combat this evil from the medical, social, and moral point of view, and with good results.

In conclusion one may add with Professor Fournier, of Paris, in imitation of another proverb, "that the fear of syphilis is the beginning of wisdom," and re-echo that fine thought of Pasteur's, "that where good is to be done duty only ceases when we no longer have power to achieve more or to do better."

Mr. McDONAGH: During recent years much legislative ingenuity has been expended on the continent by the state control of prostitution. The result has been disappointing, for syphilis seems to have increased on the continent. The reason for this is a simple one. While state control has undoubtedly rendered syphilis less common amongst professed prostitutes, it has increased the number of unregistered and therefore uncontrolled women, among whom syphilis has become the rule rather than the exception, and for such an act to bear fruit, syphilis in men should be equally notifiable. Without having state control of prostitution, there seems to be absolutely no reason why syphilis should not be included under the Contagious Diseases Act.

Sero-therapy.—Sero-therapeutic measures and preventive treatment by means of injections of the serum of animals, which had previously become infected with the syphilitic virus, have proved hopeless.

Neisser injected intra-venously a syphilitic in the primary stage with the serum of a patient who had had syphilis, but with no result.

Metschnikoff and Roux, having proved, *in vitro*, that the serum of apes which had previously been infected with syphilis possessed some parasiticidal power, tried to produce a prophylactic active immunity by obtaining a virus, weakened by the passage through several species of apes, which possessed a certain natural resistance against syphilis, but unfortunately the results proved fruitless.

Kraus obtained a vaccine from the maceration of chancres, and injected it subcutaneously in the primary stage of the disease; but although Kraus's reports are favourable, other observers have failed to corroborate them.

Excision of chancre.—The treatment of syphilis should be begun the moment the disease has become diagnosed, as Mr. Lane has, in his address, so strongly advocated, since we have every means in diagnosing a suspicious sore the moment the patient's attention has been drawn to it.

Experiments proving that the inguinal glands are affected before the patient comes up for advice have made most observers look upon excision of the chancre as being unnecessary, but they fail to realise that a chancre left may act as a fountain from which fresh organisms continually flow; and that a chancre may relapse, that papules and gummata may occur at the site of the initial lesion, and starting treatment at once causes the chancre to heal over quicker, and therefore diminishes risk of infection.

I know of two cases in which the chancre had been excised after the discovery of the specific organism about two weeks after its appearance. The patients were not treated. A Wassermann's reaction was carried out every three months for two years, always with a negative result, and no further symptoms of the disease appeared. To my mind there is no doubt that when a chancre can be easily excised it should be, as I feel quite sure that the future course of the disease is affected thereby.

Treatment and Wassermann's reaction.—The influence of treatment on the Wassermann's reaction is undeniable, but the treatment must be mercurial, as potassium iodide has no influence on converting a positive into a negative reaction.

Mercury given by inunctions has far and away the greatest influence in changing the reaction, it being practically the rule after a course to obtain a negative reaction, which later may become positive, depending upon the number of courses given.

Owing to the fact that syphilis may run a mild as well as a severe course, it will be by the Wassermann's reaction that we shall be able to gauge as to whether a patient requires more treatment or not.

This is in my opinion the greatest argument in favour of the intermittent over the continuous treatment.

We have not yet arrived at the stage when one can say positively as to whether a patient should receive more treatment or not, but it is a very safe rule to go on in saying that a patient is cured when his blood gives a negative reaction three, six, and nine months after his last taking mercury.

Such a course will not increase proclivity towards getting nervous manifestations, since such diseases are noted for the amount of anti-substance present in the patient's serum.

Injections play a subordinate *rôle* to inunctions, and it may require several before any change in the reaction is noticed.

Pills hardly come under consideration except to be doomed, first, because they have little or no influence in changing the reaction; secondly, because they upset the patient, increase the intra-buccal lesions, and predispose towards carcinoma of the tongue.

Over and over again I have noticed how bad patients' mouths and throats get when they are taking pills. It is not due to a mercurial over-dose, since alleviation sets in the moment injections or inunctions are given.

While on the Continent I was struck with the fact how seldom bad tongues were seen, also carcinoma, although syphilis was so much commoner than at home.

One cannot go into a surgical clinic in London, however small it may be, without seeing several cases of bad tongues, which get worse the further pills are pushed; not infrequently patients have asked to have injections rather than pills owing to

the bad state the mouth gets into when taking the latter.

One of the greatest arguments in favour of treating the patient as early and as vigorously as possible is shown by the fact that when a case has progressed to a certain stage, to produce some nervous manifestation or general arterios-clerosis, however vigorous the treatment may now be, it is impossible to convert a positive Wassermann's reaction into a negative one; therefore treatment at this stage can never cure the patient, only arrest the further progress of the disease, and as the lesions are due to the syphilitic virus it is sufficient indication to give mercury and not rely solely on potassium iodide, as is so frequently done, with the exception of general paralysis of the insane and tabes, conditions which are not infrequently aggravated by the use of mercury.

Arsenical treatment.—Anæmic and tubercular folk who subsequently contract syphilis are often unable to stand mercury. Mercury makes them weak and more anæmic; when resource should be had to arsenic, which is a valuable drug in any stage of the disease.

Atoxyl and other arylarsonates have been vaunted as ideal preparations, but I have witnessed two cases of temporary blindness and one of persistent headache following their use; they are, therefore, not to be recommended to any practitioner who has to live upon his reputation. It must also be remembered that these organic arsenical preparations so affect the system that a repetition of their use some months later may give rise to most alarming symptoms, owing to the patient having been made over-sensitive by their action—a condition analogous to the serum disease. As one can test the over-sensitiveness to tuberculin by means of the Pirquet reaction, the same test can be applied to drugs, and by this means one could pick out those who gave a cutaneous reaction with the arylarsonates.

A new drug, called Ehrlich-Hata's preparation, No. 606, which is dichlorhydrate, dioxdiamido-arsenobenzol, has just appeared, which is said to bring a most wonderful change in syphilitic lesions at any stage of the disease after one injection of gr. v.

Hata has already used it in Frankfurt in more than 100 cases of syphilis without meeting with any untoward symptoms. Out of twenty-five patients reacting positive twenty-three gave a negative Wassermann after one injection.

Syphilis in mother and child.—Owing to the fact that a woman who aborts or bears syphilitic children is herself immune, because she is syphilitic, she should be treated, whether she shows symptoms or not, throughout the whole time of each successive pregnancy, with the hope of begetting a normal child. It is important also for the father to receive treatment should he give a positive Wassermann's reaction. A child born of a suspected syphilitic mother should, if a positive Wassermann be obtained, be treated for at least two or three years. No hard and fast line can be drawn as to how long treatment in such cases is necessary, owing to the fact that congenital syphilitics give a positive Wassermann, whether under the influence of mercury or not. Too much stress cannot be laid upon the importance of testing a woman's blood who aborts or has premature labours. In drawing up the statistics on infant mortality, it will be noticed how small a *rôle* syphilis seems to play, for the simple reason that more congenital syphilitics are born dead than alive. Hence it is so important to get every syphilitic woman under treatment as soon as possible. A child born of syphilitic parents may show no signs until some time after birth; therefore all children born to suspected parents should, whether they show symptoms or not, if they give a positive Wassermann, be treated with mercury, since by so doing we shall be able to avoid the horrors of juvenile tabes, double interstitial keratitis, etc.

Hochsinger, since 1869, has been able to keep under supervision 134 women, who showed no signs of syphilis, but begot syphilitic children. These women gave birth 569 times, out of which 253 were born dead—44·4 per cent.; 263 were syphilitic, and 53 were without a taint. Of the 263, 55 died before the fourth year—over 20 per cent.

That treatment of the parents has an enormous influence on the severity of the disease in the offspring, is shown so well in private cases, where the parents are more systematically treated, and the number of syphilitic children are fewer and the disease less severe.

(*To be concluded.*)

June 27th, 1910.

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CONTENTS.

PAGE

***A Clinical Lecture on Two Cases of Carcinoma of the Rectum Treated by the Abdomino-perinæal Method.** By H. J. WARING, M.S., F.R.C.S. ... 193

***A Clinical Lecture on Asthma.** By A. F. HERTZ, M.D., F.R.C.P. 198

***Syphilis.** A discussion by Mr. ERNEST LANE, Mr. ALEXANDER FLEMING, Major FRENCH, R.A.M.C., Mr. MCDONAGH, Mr. C. F. MARSHALL, Dr. PERNET, Mr. H. W. BAYLY (*concluded*) 205

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A CLINICAL LECTURE

ON

TWO CASES OF CARCINOMA OF THE RECTUM TREATED BY THE ABDOMINO-PERINÆAL METHOD.

Delivered at St. Bartholomew's Hospital.

By H. J. WARING, M.S., F.R.C.S.,

Surgeon to the Hospital, and Consulting Surgeon, Metropolitan Hospital; Examiner in Surgery, University of London.

GENTLEMEN,—I regret that I have had to make a change in the title of my lecture which I announced last week. The patient about whom I had hoped to speak and to show to you is not available. I shall therefore discuss two cases of carcinoma of the rectum treated by the abdomino-perinæal method.

With regard to the first case, the patient was a woman, æt. 50 years, who came to the hospital complaining of passing blood in her motions, and of having occasional attacks of diarrhœa. Otherwise she appeared to be, and felt, fairly well. On examination she was found to have an indurated ulcer situated in the wall of the rectum and commencing four to five inches above the anal orifice. This involved the entire circumference of the bowel, extended beyond the reach of the finger, and so narrowed the lumen of the gut that the finger could not be passed through and beyond the diseased area. On bimanual examination the growth in the rectal walls could be distinguished as a tumour projecting towards the supra-pubic region, which was freely movable, both from side to side and also to some extent from above downwards. The diagnosis was made that the patient had carcinoma of the upper portion of the rectum, and mainly involving the part covered by peritoneum. Further investigation of the patient did not show that she suffered from any secondary

growths; there was no evidence of enlarged lymphatic glands, either in the hollow of the sacrum or in front of the spinal column, or of enlargement of the liver. So from the point of view of a patient suffering from rectal carcinoma, I concluded that it was one of those cases for which complete excision was the best treatment. I shall revert to the particular kind of excision shortly. There were one or two other clinical points in connection with the patient which made it advisable to discuss the case. She was stated to have tuberculous disease of the lungs, one lung being extensively diseased, and also varicose ulcers on one leg. However, I asked my medical colleague, Dr. Tooth, to examine and report upon the condition of her lungs, and he said there was no active phthisis there now, though at the apex of one lung there were signs of an old cavity. With regard to the varicose ulcers of the leg, rest in bed and the application of local antiseptic measures enabled us to get them into a satisfactory condition. Having decided that complete excision was advisable, it was then necessary to discuss the form of operation which should be done in her case. I shall now send round this specimen removed at the operation for your inspection, because I wish you to realise the position, extent, and connections of the growth. First of all I ask you to notice the anus, which is at the bottom; secondly, the portion of intestine which is not covered by peritoneum; thirdly, the growth; fourthly, the portion of intestine above the growth; and fifthly—which is very important—various enlarged lymphatic glands, which are dissected out and lie between the two layers of the meso-rectum. You will now be able to follow the chief points as I proceed in regard to treatment. I recommended this patient to have the entire growth removed by the abdomino-perinæal method, since by this operation I should be able to excise the involved portion of the rectum, together with the parts above and below, and also the groups of lymphatic glands which receive the lymphatic vessels from the diseased area. That is the main object which one tries to carry out as far as possible in the treatment of malignant disease anywhere, especially if that malignant disease be carcinoma. The operation which I performed upon this patient I will now describe to you in some detail. There is one point, however, I wish to refer to in connection with this case, so as to clear the way. With regard to all cases of excision of the rectum by the abdomino-perinæal operation, there is one question which always arises—whether it is desirable before the actual operation of excision is carried out to perform a preliminary inguinal colostomy. Opinions are very divided among surgeons as to whether that should be done or not. My own view is that when a patient presents no definite signs of acute intestinal obstruction, and it is possible by means of aperients to clear out the entire alimentary canal, so as to leave no accumulation of fæcal material above the narrowed portion; then, as a general rule, a preliminary inguinal colostomy should not be done. You are likely to get a better result by not doing it. But if, on the other hand, the carcinomatous growth has so extended that the lumen of the gut is encroached upon and narrowed, and there is an accumulation of intestinal material above, and one cannot by aperients evacuate the large intestine, then in those cases I think it is very advisable before the excision operation is performed to carry out a preliminary left inguinal colostomy. About a fortnight should be allowed to elapse before performing excision, so as to permit a thorough evacuation of the bowel, and periodical washing out of the portion of intestine which lies below the colostomy opening. This is a very important consideration in connection with these cases, since when there is obstruction there is usually decomposition of the retained fæcal matter and absorption of the toxic products. In this particular patient there was no acute obstruction and retention of fæcal matter, hence preliminary inguinal colostomy was not necessary. The operation which we performed on her was, roughly, as follows: The patient, after being anæsthetised, was placed in what may be called the *high* Trendelenberg or raised pelvic position. It is very important, with regard to this operation, that the pelvis should be well raised, so as to enable one to pack the small intestines and the great omentum upwards towards the diaphragm, and so leave the rest free, with the exception, of course, of the genito-urinary organs or the pelvic portions of them, and that portion of the rectum which has to be dealt with. The first stage comprises: opening the abdominal cavity through a median incision, six inches in length, in the hypogastric region, and packing away the intestines and omentum, so as to cut off the portion of the abdominal cavity which

lies above the pelvis. When this has been done it is necessary to provide a thorough exposure of the rectum. This can most readily be effected by using a large Doyen's retractor of the shape I show you. The essential part of this instrument is a very large retractor, which is placed transversely across the wound, in its lower part, so as to separate the margins below and lay bare the pelvic contents. After it has been inserted it is held in position either by a dresser, who holds it during the first part of the operation, or when such a dresser is not available, placing the thigh piece between the patient's thighs and clamping it in that position. For pelvic operations of this kind it is exceedingly useful. When this exposure has been made one examines carefully the pelvic contents in order to confirm the diagnosis, and to ascertain the mobility of the diseased bowel and the possibility of its local removal. Secondly, the meso-rectum and meso-sigmoid, or the meso-pelvic colon are investigated in order to determine the condition of the lymphatic glands between their layers, since these lymphatic glands receive lymphatic vessels from the portion of the rectum which in this case was involved. Investigations in this case showed the presence of a number of hard and distinctly palpable lymphatic glands lying between the two layers of peritoneum. In the specimen you have seen the peritoneum has been taken away and the lymphatic glands left behind, so as to show their position, the extent of their enlargement, and how they were removed. The operation being considered locally feasible, the next thing to do is to pass the hand upwards into the abdomen and palpate the liver, and also the chain of lymphatic glands in front of the spinal column, in order to exclude the presence of secondary growths in these structures. No growths having been discovered in these situations, the peritoneum on each side of the pelvis posteriorly and round the floor and anterior wall of Douglas's pouch is divided, so as to free entirely the meso-rectum, the pelvic portion of the colon with its mesentery, and that portion of the rectum which lies below. A further separation of the part of the rectum which lies below, and which is uncovered by peritoneum, is done chiefly with the fingers or blunt dissector, after the blood-vessels which lie in the mesentery and meso-pelvic colon have been clamped and cut through. The part of the rectum below can be readily separated from the surrounding and adjacent structures as far as the upper aspect of the pelvic diaphragm—that is to say, the pelvic fascia covering the levator ani muscles. This completes the first main stage of the operation: exposure and separation of the rectum and the pelvic colon from above, and clamping and ligature of the blood-vessels in the meso-pelvic colon. When this has been finished the margins of the separated portions of the rectum are packed with a couple of flat pads, and then the wound in the hypogastric region is temporarily closed by salmon-gut sutures passed through the entire thickness of the abdominal wall. An aseptic dressing is next applied and fixed in position preparatory to the next stage of the operation. The patient is now lowered from the raised pelvic position and placed lying on the left side. Then the anal and perinæal regions, if not already done, are cleaned up and made aseptic. The mucous membrane or muco-cutaneous junction at the margin of the anus is cut all the way round, dissected up for a short distance and tied with a silk ligature, and then covered with gauze to prevent contamination. The mucous membrane of the anal canal is dissected upwards for one and a half inches, leaving behind the sphincters. A further incision is made backwards towards the tip of the coccyx, dividing the ano-coccygeal ligament, thus fully exposing the anal canal. The floor of the pelvic diaphragm behind is separated further until the pelvic cavity is reached, and then with the fingers the entire rectum is brought out through the perinæal wound. During this dissection a limited amount of hæmorrhage is met with, but it is not considerable, because the superior hæmorrhoidal vessels have already been tied above, and this prevents serious hæmorrhage below. The separated rectum, including the part in which the growth is situated, is now brought out through the perinæal wound, together with the lower portion of the pelvic colon as far as it has been separated, the gut being brought down until sound tissues well above the region of the growth and the affected lymphatic glands in the mesentery are in view. The walls of the pelvic colon are next fixed to the margins of the perinæal wound by careful suturing, beginning at the levator ani muscles above, and extending downwards as far as the region of the anus. Then the wound behind is closed, except for a small

portion where a drainage-tube is inserted. Then the patient is again placed in the dorsal posture after temporary dressings have been placed in the region of the perinæum. The abdominal wound is now opened up, the protecting pads on the floor of the pelvis are removed, and the divided margins of the parietal pelvic peritoneum are fixed by sutures to the sides of the pelvic colon. In this manner to a large extent the peritoneal pelvic diaphragm is reconstituted, only at a slightly higher level. There is some dead space left posteriorly, but this is dealt with later by a drainage-tube inserted in the posterior angle of the perinæal wound. The suturing having been completed, the wound in the abdominal parietes is closed by three tiers of sutures, namely peritoneum to peritoneum, muscle to muscle, and skin to skin. The dressings are now applied, the patient is again turned on to the side, and the portion of bowel below the anal margin is cut across with the knife of a Paquelin's cautery, and the cut margins are fixed to what was formerly the margin of the anus by several sutures, and a rubber tube of large calibre is passed into the bowel, and dressings applied. The operation is thus completed, and, as you will have gleaned, it is an extensive and complicated one, but for this variety of case, where the patient is suffering from carcinoma of the rectum and the lymphatic glands are involved, and where the growth is situated in the position I have mentioned, it is the only feasible operation which offers the patient a fair chance of a good permanent result being obtained. I have already told you that there was a small dead space left below the newly constituted pelvic diaphragm in the hollow of the sacrum. After the posterior portion of the perinæal wound has been closed a rubber tube is passed through the posterior angle of the wound into this space, and left in for a few days. This cavity, or dead space, may readily become filled with a collection of blood and serum, and be the seat of suppuration.

The after-treatment is as follows: The patient is kept absolutely quiet for a few days on a light liquid diet, and small doses of opium are given to prevent the bowels acting. At the end of five days an aperient is given so as to clear out the intestines, and afterwards the perinæum has to be periodically dressed when there is an action of the bowels. This completes the details of the first case to which I wished to draw your attention.

The second patient's case was practically a repetition of the first. She was an unmarried woman, æt. 43 years. The growth was situated a little higher up, and she had not the two complications I have referred to above, namely, an affection of the lung and an affection of the leg. The only pathological condition we found in her was that of the rectum, and an operation was carried out upon her similar to that which I have already described.

The result in well-selected cases, as far as my experience goes, in abdomino-perinæal excision of the rectum by the method already described, is that one is enabled to very effectively remove the disease, and hence offer the patient an excellent prospect of cure. A certain number of patients so treated, however, suffer for a time from incontinence or imperfect control of fæces. They do not regain that control of the motions which one would rather expect them to have. With regard to these two patients, the older woman has now, eleven weeks after operation, practically complete control, but the other patient, thirteen weeks afterwards, has not as yet. She is improving, however, but she is not in a condition in which it will be advisable for her to follow her ordinary occupation. [When seen two months later this patient had regained fair control.]

What are the facts of most importance that we can learn from these patients? The first is that it is absolutely essential for us to realise the great importance of being able to make an early diagnosis of the patient's disease, because upon that, I think, rests entirely the possibility of being able to do an operation such as I have suggested. If a carcinoma of the rectum is allowed to grow, to become adherent, or extensively adherent, we will say, to the adjacent pelvic viscera, then this operation is out of the question. Hence it is very important, from a practical point of view, that you should realise first of all the risks of a patient who is suffering from this disease, and secondly, the very great necessity of recognising the disease at as early a stage as possible. I mention this particularly, because patients who suffer from carcinoma of the rectum very frequently suffer, or have suffered, from piles, and when they first come under observation they complain very often merely of piles. Piles is a clinical condition which they have heard something about, and which they

generally think they know something about. When you first examine the patient you may find that he or she has piles, that is to say, there is a varicose condition of some of the veins in the lower part of the rectum and anal margin. They may not seem very serious, and you therefore decide to treat them by palliative measures without examining the interior of the rectum. The mistake which is made is this: Men very often forget, or omit, to examine a patient *per rectum* when examining him for *rectal symptoms*. If you are not going to pass cases of carcinoma by, you should make it a rule whenever patients come under observation and they complain of piles or the passing of blood *per annm*, or with the stools, never to omit to make a complete digital examination of as much of the rectum as can be investigated with the finger. Also in those cases where there is nothing abnormal to be made out in the form of piles, and the digital examination reveals nothing pathological, and there is still evidence of the patient passing blood *per anum*, or having occasional attacks of diarrhœa, followed by the passing of blood or slime, do not be content with merely investigating by the finger from below, but have the patient anæsthetised and have the upper portion of the rectum and lower portion of the pelvic colon examined by the sigmoidoscope. If you do this you will not miss a case of carcinoma in the upper part of the rectum, but if you do not examine these cases in that way you are very likely to miss them. Thus one important conclusion which we can learn from these cases is that, if the radical operation is to be carried out for the cure of carcinoma of the rectum, a diagnosis must be made at a comparatively early stage, otherwise the disease will be so extensive, or will have given rise to so much secondary disease in other parts, that operation will be out of the question.

Another point of interest which I wish to discuss in connection with this condition is the advisability or non-advisability of inguinal colostomy, either as a preliminary procedure or what may be called part of the main operation. In the first patient it does not appear that inguinal colostomy would have been of any value, because she can now retain her motions and pass them in the usual way, or at all events by the usual route. In the second patient, I think the question may possibly arise as to whether it will not be advisable to consider, if she completely fails to get control, whether an inguinal colostomy should not be done. This I understand is the experience of most surgeons who have had many of these cases to deal with, that a certain proportion of the patients for whom this operation is done have incontinence of fæces afterwards. That is, of course, a very unsatisfactory condition, and it is a condition which is not very readily managed by any appliance. There are no appliances which are very efficacious in making a patient comfortable who has incontinence of fæces. When that occurs, after the rectum has been excised for carcinoma, as far as my experience permits me to form an opinion I think far better results are obtained by performing inguinal colostomy. This being admitted, a number of surgeons say that it is far better, as regards this operation, to do an inguinal colostomy to begin with, and not to make any attempt to take the intestine down to the anal margin and fix it there. When the abdomen has been opened and the pelvic viscera exposed, they divide the large intestine well above the primary disease and the portions of the mesentery which contain the affected glands. They sew up the lower extremity of the upper upper segment, and then perform inguinal colostomy. And then the segment of intestine which lies below is excised in a manner similar to that I have described, and the perinæal wound is closed except for a small part in which a drainage-tube is placed. This allows any material to pass out, and ultimately, of course, this tube is taken out, and there is no pelvic exit at all. The question of colostomy is a point as regards these cases which at the present time may be said to be under consideration, and I do not know that there is a sufficient number of cases which have been reported and watched for long enough to enable us to say which is really the best procedure. I am of opinion, however, that when there is no acute obstruction inguinal colostomy ought not to be performed, since an intestinal outlet in the usual position is far preferable to an inguinal one. During this lecture I have been dealing with cases of carcinomata in which the disease is situated, as it not uncommonly is, in the position shown in the specimen.

As regards carcinoma of the rectum in general, there are several points to which I would draw your attention. Here is a specimen to illustrate the condition which I mentioned to you, when a

patient has acute intestinal obstruction caused by the new growth. It is a carcinoma of the rectum in which the lower part is comparatively narrow, the middle part is occupied by an annular ring, or thickening of new formation, and the part above is considerably dilated. Whenever you find this condition of affairs it is not advisable to even consider the operation which I have been discussing. You must perform in these cases preliminary inguinal colostomy. Carcinomata of the rectum are often divided, as regards seat, into three groups: Those which occupy the lower part, that is to say, anywhere between the anal margin and three to four inches above. The carcinomata which occupy this portion of the rectum are often of the adeno-carcinomatous type; they form a considerable amount of growth, which involves the interior of the bowel, and as the lumen of the bowel there is of large size there is no acute obstruction. The second group of cases are what may be called the annular, and many of them occur in the portion of the rectum above the lower three inches. Those are the cases which are especially suitable for the particular operation I have referred to. There is a third group of cases, of which I have another example here, and that is where a considerable segment of the rectal wall is involved by the growth. Here four inches of the rectal wall is involved by a growth which, histologically, has undergone colloid degeneration. The most favourable cases for operation are the annular ones, which involve the portion of rectum above the lower three inches, and of course within the pelvis.

July 4th, 1910.

In the revised edition of Bland-Sutton's 'Position of Abdominal Hysterectomy in London,' just issued by Nisbet & Co., 22, Berners Street, W., the author says that since 1894 he has performed more than a thousand operations of various kinds for fibroids, and has lost one patient from pulmonary embolism. That happened in 1900. Probably no amount of care will absolutely abolish the liability of patients to pulmonary embolism after pelvic and abdominal operations, but the thrombosis, which is not only a serious complication in itself, but the forerunner of the graver embolism, is often due to inefficiency in the means taken to procure asepsis. As Mr. Bland-Sutton very truly says, it is a misnomer to speak of the "uncontrollable embolus."

A CLINICAL LECTURE

ON

ASTHMA.

Delivered at Guy's Hospital.

By A. F. HERTZ, M.D., F.R.C.P.,
Assistant Physician to the Hospital.

Gentlemen,—Asthma may be defined as a condition in which there are paroxysmal attacks of expiratory dyspnœa, associated with defective respiratory movement with no constant organic lesion, and compatible with good health between the paroxysms.

In considering the pathology of asthma, it is necessary to discuss three different things: first, the condition of the bronchi; secondly, the condition of the central nervous system; and thirdly, the exciting causes of the attacks.

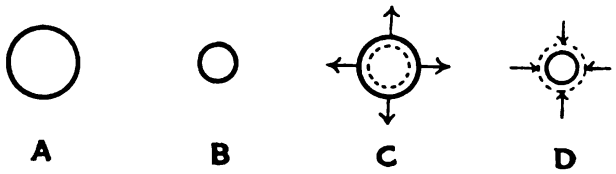
(1) *The bronchi.*—In a typical case of asthma there is over-action of all the structures of the bronchi—spasm of the bronchial muscles, congestion of the bronchial mucous membrane, and over-secretion of the bronchial mucous glands. On stimulating the vagus nerve of an animal contraction of the bronchi occurs, and there is no doubt that in asthma the spasm of the bronchial muscles is caused by impulses carried by the vagus. The effect of the spasm on the bronchial muscles is to diminish the quantity of air which can pass into and out of the lungs. In this connection the difference in the condition of the bronchi in expiration and in inspiration is of great importance, because the most characteristic feature of bronchial asthma is that the dyspnœa is an expiratory dyspnœa. In order to explain this I have drawn these diagrams (Fig. 1). The circle A represents the size of a bronchiole under normal conditions, and B its size during an attack of asthma. In inspiration the chest enlarges, and as a result of that the bronchioles are pulled open (C), and the air can enter the lungs without much difficulty. On the other hand, during expiration the increased intra-thoracic pressure compresses these bronchioles so as to make their lumen smaller (D). During expiration there is thus considerable obstruction to the passage of air out of the alveoli, and consequently there is expiratory dyspnœa. It

is comparatively easy for air to be drawn into the lungs, but difficult for air to be expelled; the lungs become greatly distended, the distension being only slightly diminished during expiration.

In addition to the spasm of the bronchi, there is congestion of the mucous membrane. It is generally present in the nose also, and it has been seen by means of the laryngoscope in the trachea and in the upper part of the two main bronchi, so that it is probable that this congestion extends all the way down to the smaller bronchioles. It is simply the ordinary accompaniment of functional activity in a muscular tube.

Lastly, there is generally increased bronchial secretion. In some individuals attacks of pure spasmodic asthma may occur with little or no secretion, but in the majority of cases there is a considerable quantity. The secretion of the bronchial mucous membrane, when there is no accompanying bronchitis, is entirely mucus. Owing to

FIG. 1.



the spasm of the bronchial muscles there is considerable difficulty in expelling the mucus; instead, therefore, of being expectorated more or less rapidly as in ordinary bronchitis, it remains locked up in the smallest bronchioles. Owing to the action of a ferment—mucinase—contained in the bronchial mucous membrane, it becomes to some extent coagulated. The coagulated mucus is slowly expelled through the bronchioles, the structure of which is spiral, so that it assumes the characteristic form of Curschmann's spirals. In all cases of asthma uncomplicated by bronchitis, in which any sputum is brought up, you can find shreds of mucus, which are casts of the smallest bronchioles, and in which you can see with the naked eye a spiral structure. Another characteristic of the expectoration is the presence of Charcot-Leyden crystals and an excessive number of eosinophile leucocytes. The former are produced by the latter, so that wherever eosinophile leucocytes are in excess Charcot-Leyden crystals are also found. Associated with the formation of eosinophile leucocytes by the bronchial mucous membrane an increase of eosinophile corpuscles occurs in the blood so long as the attack lasts.

(2) *The central nervous system.*—We now come to the part played by the central nervous system in the production of asthma. In all cases of asthma there is an abnormal excitability of that division of the respiratory centre which controls the activity of the bronchial muscles. Many cases of asthma are hereditary, several members of a family suffering from the disease; what is inherited is probably this abnormal excitability of the bronchial centre. Frequently there is no history of asthma in the family, but several of the patient's relations suffer from various neuroses; in such cases the unstable nervous system is inherited and manifests itself in the over-excitability of this centre. Whenever the centre governing the activity of the bronchial muscles is abnormally excitable, any stimulus which reaches it will produce an exaggerated response, and will therefore be liable to cause an attack of asthma.

(3) *Exciting causes.*—The irritable bronchial centre may be stimulated by impulses coming from the higher parts of the brain, and by reflex impulses. The former occur particularly when a predisposed individual is under the influence of excitement. Thus I have known students who had a severe attack of asthma before every examination. Asthmatic subjects are particularly liable to attacks in certain places; having once had an attack of asthma in a certain town, they are exceedingly likely to get an attack if they go there again, not only because the atmospheric conditions which produced the first attack are present still, but also because they expect to get another attack there. A patient who had great faith in a certain remedy had an attack every night; he was far worse if he forgot to take it with him, although he rarely required it if he did take it. A lady had an attack of asthma every time she smelt a rose: this was proved to be largely a psychical process, as she had a severe attack on seeing some artificial roses, which she took to be genuine.

The most important group of peripheral stimuli which give rise to attacks of asthma reflexly are those connected with the respiratory tract itself. Mechanical or chemical stimulation of the posterior part of the nasal septum produces reflex bronchial spasm in animals. In asthma the reflex generally

arises by stimulation of the nasal or bronchial mucous membrane by certain atmospheric conditions. Attacks are particularly common in close and damp weather, but they depend more particularly on less easily defined conditions. Every asthmatic will tell you that he knows certain places in which he is liable to attacks, and others in which he is comparatively free from them. But a place which is good for one patient may be the worst possible place for another. In my own case I have been quite unable to find what are the climatic conditions which give rise to an attack. I have never had asthma in London, but have had it at Streatham; I have never had it in Manchester, Leeds, or Sheffield, but have had it in Bradford; I have never had it at Oxford, but have had it at Stratford-on-Avon; I have never had it at various seaside places, such as Llanfairfechan and Worthing, but at others, such as Criccieth and Littlehampton; I have had it on the Mediterranean and the North Sea, but not on the Atlantic; and in Strassburg and Heidelberg, but not in Munich, Berlin, or the Black Forest; I have never had it in the Alps, in Spain, or in Paris. In a motor tour from London to Land's End and back I had it for a few hours while going and coming back whilst in Salisbury, but at no other place.

London is the best place in England for most asthmatics, who may never get an attack unless they go into the country. It seems clear, too, that the more smoky part of London is very much better than the suburbs. Very slight changes in the atmospheric conditions may be sufficient to give rise to an attack. Thus many asthmatics find that they can live in one street, or house, or room, but not in a neighbouring one. I have spent some 200 nights in Guildford in various rooms in six different houses, but have only twice had asthma there, both attacks occurring on the only two occasions I slept in a certain room, although several months elapsed between the two attacks, and I never thought of the first attack on the evening on which the second attack occurred.

In addition to general atmospheric conditions, certain odours and emanations cause attacks. Some patients get asthma when pollen is in the air, although this more frequently leads to hay fever. The smell of ipecacuanha produces an attack of asthma in many asthmatics, and there are numerous well-authenticated cases of attacks being produced by the emanations of certain animals, especially of cats. One example of "cat asthma" which I have myself come across was that of a lady, in whom the attacks did not depend on her actually seeing the cat, so that it was clearly not a purely neurotic condition. She could always tell whether a cat was in the room by the feeling of dyspnœa which came over her. On one occasion, while paying a visit to a friend whom she knew kept no cat, she said to her hostess, "There is a cat in the room, as I am feeling asthmatic"; it was only after a search that a stray cat was found hidden behind a curtain. I have come across one case of asthma caused by the emanation from horses. A farm labourer, who had never had an attack of asthma, and whose work had never brought him into close contact with horses, had on one occasion to do the work of the man who was accustomed to drive the cart containing the farm produce to the nearest market. For the first time in his life he sat behind a horse. As he was driving along he gradually became asthmatic, and by the time he reached the town, which he had often visited before, he had to be lifted from the cart because of the severity of his asthma. Later on he obtained a situation as a groom—a very unwise thing to do; while in that situation he constantly had asthma, until he discovered for himself that the wearing of a cloth mask over his nose and mouth prevented the attacks. I saw the mask which he was in the habit of wearing, and so long as he wore it he remained perfectly well.

It is not only the bronchial centre which is abnormally excitable in asthmatics. In some patients part of the peripheral respiratory mucous membrane is also abnormally excitable, and so the patient is in two ways predisposed to attacks of asthma. This occurs particularly in cases in which the nasal mucous membrane is abnormal, the turbinals being hypertrophied, or the septum deflected. Much more rarely nasal polypi or adenoids are the cause. In the presence of these abnormalities, the mucous membrane becomes abnormally excitable, and a patient who has also an abnormally excitable centre becomes liable to attacks originating in the nasal mucous membrane. In such cases considerable improvement will ensue from treatment of the nasal mucous membrane.

After the respiratory reflexes, the most important are those which arise from the alimentary canal.

Particularly in children, it is very common for an attack of asthma to originate from gastric irritation. In some cases special articles of diet will produce an attack of asthma; the patients usually discover that for themselves. But more commonly simple distension of the stomach by a bulky meal is the exciting cause. In some asthmatics every meal, unless it is very small, and especially in the evening, gives rise to some dyspnœa, if not actually to asthma. This is often associated with aërophagy, and the flatulence produced thereby greatly increases the feeling of dyspnœa. An attack of asthma produced in this way can often be cut short by vomiting. It is doubtful whether asthma ever arises from irritation of the intestines, with the exception of the pelvic colon and rectum. An accumulation of fæces in these parts will in many cases produce dyspnœa in the predisposed. One of the most important considerations in the treatment of asthma is to see that the bowels are opened regularly. Some patients find that when they have an attack of asthma an enema will give immediate relief. The two worst attacks of asthma I have ever seen were suddenly terminated by the passage of a copious watery stool.

A less common reflex cause is from the genital organs. There are some individuals in whom pregnancy has produced attacks of asthma, and I know one case in which the removal of a fibroid of the uterus apparently brought permanent relief from asthmatic attacks.

The last source from which asthma may arise reflexly is the skin. It is not uncommon to find asthmatics who suffer also from various skin diseases, such as eczema and urticaria; most commonly the attack of asthma comes on when the skin condition is improving. Whether this is due to a reflex is not clear, but there is no doubt that in some individuals sudden cold applied to the skin will give rise to an attack.

Lastly, it is probable that chemical stimuli can act on the irritable centre; perhaps the poisons which are associated with gout act in this way, because gouty people are specially prone to asthma. The dyspnœic attacks which occur in uræmia may be partly due to bronchial spasm brought about by the action of the uræmic poisons on the centre, so that the expression "uræmic asthma" may in some cases be justified.

Symptoms.—It is not generally recognised that many attacks of asthma are preceded by a definite aura, which may be present for twelve or more hours. There may be a feeling of depression, or some abnormal cutaneous sensation may be noticed. One patient felt as if a burning iron was being rubbed over his back, and another always experienced an itching sensation before an attack. Most frequently there is simply a feeling of tightness in the chest, which is perhaps really the beginning of an attack rather than a genuine aura. Attacks of asthma commonly occur in the night. The reason for this is not very clear, but it depends to a great extent on the recumbent posture which is ordinarily assumed. The congestion of the nasal and bronchial mucous membrane, which occurs when the patient lies down, appears to make the mucous membrane more irritable.

Asthmatic patients often go to sleep normally, but at about 2 o'clock in the morning they wake up with a feeling of great distress. As I have already said, the characteristic feature about an attack of asthma is the expiratory dyspnœa. Swift makes Anthony Henley's father, dying of asthma, say, "Well, if I can get this breath once out, I'll take care it shall never get in again." The speed of respiration is not increased, but expiration is prolonged to three or four times its normal duration, and there is a feeling of very great distress, the difficulty in getting rid of the air from the lungs leading to powerful expiratory efforts. On examining the chest one finds that it is expanded to the greatest possible degree. By means of the X-rays the diaphragm can be seen to be fixed in a position as low down as it can go. This is generally regarded as a simple mechanical result of the inflated condition of the lungs, which results from the bronchial spasm. But it is very frequently associated with a sensation of aching at the attachments of the diaphragm, which may last for several hours after the attack has disappeared. From my own sensations I am convinced that spasm of the diaphragm occurs in addition to the spasm of the bronchi. During an attack the inflation of the lungs causes the cardiac and hepatic dulness to be greatly diminished. The thoracic movements are exceedingly small, in spite of the great straining during expiration. On listening to the patient's chest one can hear rhonchi, which are much more marked during expiration than during inspiration. Generally they are evenly distributed all over the

chest, and they are often so loud that they can be heard at a distance from the patient. But in the type of case in which spasm is the only marked feature, and in which there is very little secretion, there may be comparatively few rhonchi.

The attack usually lasts an hour or two, after which, even if it is not treated, it slowly passes off and the patient frequently falls asleep again. As the severity of the attack diminishes, the patient often coughs up mucous fluid, containing Curschmann's spirals; their expulsion helps to give relief, as they probably cause some obstruction of the bronchioles.

During the whole of the day following an attack of asthma the patient feels slightly dyspnœic and his breathing is wheezy. He cannot go upstairs without feeling very uncomfortable, and if he takes an ordinary-sized meal he will at once feel short of breath. The dyspnœa usually passes off to some extent in the late afternoon, but it is present more or less all through the day. So long as the conditions which gave rise to the first attack remain, an attack will occur every night and the slight dyspnœa will be present every day. In very severe cases there may never be complete freedom, but the majority of patients are fairly well during several months every year, and only under special conditions and in special places will they become asthmatic, with nocturnal attacks and discomfort during the day. Sometimes after a series of attacks a patient may remain slightly asthmatic for several days or even weeks, although he may sleep well and have no true asthmatic attacks. The dyspnœic condition, which is present without attacks, or in the intervals between attacks, may be termed the *status asthmaticus*.

Diagnosis.—There is no great difficulty in diagnosing asthma if it is remembered that asthma is almost the only condition in which there is expiratory dyspnœa with no increase in the rate of respiration. Another characteristic feature is the expanded condition of the thorax and the small respiratory excursion. In both laryngeal and tracheal obstruction the dyspnœa is inspiratory and not expiratory, and in these conditions the violent movements of the larynx and the peculiar stridor are characteristic. In bronchitis there may be nocturnal attacks of dyspnœa, due to accumulation of secretion in the bronchi, which closely resembles asthma. But whenever the dyspnœa is definitely expiratory, and the nocturnal attacks begin and end somewhat suddenly, there is probably a true asthmatic element in the case, the local irritation produced by the bronchitis leading to a secondary reflex spasm of the bronchi. In the so-called attacks of uræmic and cardiac asthma, respiration is much more rapid, the dyspnœa is not expiratory, and the condition of the urine and of the heart suggests the correct diagnosis. In the so-called hysterical asthma respiration is extremely fast and there is no genuine dyspnœa at all; the condition should therefore be called hysterical tachypnœa and not asthma.

Prognosis.—The danger in an actual attack of asthma is very small indeed. I only know of three recorded cases of death during an attack. One almost fatal case occurred in Guy's many years ago: a student was taken into Clinical with a very severe attack of asthma; during the night, the house-physician, who in those days slept in an adjoining room, was hastily summoned on account of an alarming failure of respiration; breathing became more and more shallow and finally ceased. The patient then fell forward in a state of unconsciousness. Artificial respiration was at once performed, and after several minutes consciousness returned. There is little doubt that death would have occurred if medical aid had been less prompt. As a rule, serious consequences are prevented by the severest attacks being brought to an end by the exhaustion of the irritated centres, so that the spasm relaxes and the patient falls asleep. Moreover, in extreme cases, the spasm can always be relaxed by the use of morphia or chloroform.

A patient, who is subject to asthma, will not necessarily always remain subject to attacks. The fewer attacks he has and the more he does to prevent having attacks, the more likely he is to lose his liability to them. The less the irritable centre is stimulated, the less irritable it becomes, until finally it may cease to be abnormal at all. It is not uncommon for asthmatic children to grow out of it entirely.

The only complication of asthma which must be considered is bronchitis. This always develops in patients who are continually asthmatic, with no real interval of complete freedom. It is partly a result of the irritation caused by constantly recurring attacks, but very often it is due more to injudicious treatment by the irritating fumes, which

are commonly inhaled by asthmatics. When chronic bronchitis has once developed, it will lead to emphysema and right-sided heart failure, and the patient may finally die from the chronic bronchitis, emphysema, and heart failure, which have complicated his asthma.

Treatment: (*a*) *The attack.*—In the treatment of an attack of asthma, attention must be directed to the bronchi, the central nervous system, and the exciting cause.

(1) *The bronchi.*—An attempt must be made to overcome the spasm. It has been definitely proved that atropine paralyses the vagal nerve-endings in the bronchi, so that atropine and the allied alkaloids are effective remedies for an asthmatic attack. In severe cases the subcutaneous injection of $\frac{1}{75}$ gr. of atropine sulphate generally produces immediate relief. A more common method of administration is by inhalation of the fumes of burning stramonium leaves, nitre being added in order to render the powder inflammable. Stramonium, like belladonna, contains atropine and the allied hyoscyamine; it is generally used in preference to belladonna for asthma, although there is practically no difference in the proportions of the alkaloids present in the two drugs. Lobelia, though it contains no atropine, seems to act much in the same way as stramonium, and it now is a common ingredient of asthma powders. The fumes produced by burning any of the numerous asthma powders, which differ little from each other in their essential ingredients, have the great advantage of irritating the bronchial mucous membrane. For a patient in an attack brought on by some temporary cause, such as a visit to a place which does not agree with him, there is no harm in using one of these powders. But their habitual use for chronic asthmatics should never be allowed, because it produces the most serious complication of asthma—chronic bronchitis. Nitrites give relief to some patients, owing to their directly paralysing action on the bronchial muscles. Every asthmatic soon learns that he is most comfortable when sitting or standing. This is largely due to the fact that he can then contract his expiratory muscles more powerfully than when lying down. But as an attack can often be prevented by going to sleep in a chair or propped in bed instead of lying down, it is clear that there must be some other factor. This is the effort of gravity on the distribution of the blood. In the vertical position the mucous membrane of the nose and bronchi is much less congested than when lying down, so that the reflex from the nose and the obstruction of the bronchi are both diminished. This can readily be demonstrated in a patient with partial nasal obstruction, as this is always worse and often becomes complete as soon as he lies down.

(2) *The central nervous system.*—In the very worst type of attack it may be necessary to give chloroform or to inject morphia, and then the attack will disappear at once. No person should be allowed to die from an attack of asthma, as when the dyspnœa is so severe that his life seems to be in danger, an injection of morphia produces immediate relief by paralysing the centre. But this is a form of treatment which should be reserved for extreme cases, as no patients are more likely to become addicted to the drug habit than chronic asthmatics. But there is a more valuable method of treating the central nervous system during an attack than by means of morphia, and that is by psychical means. If a patient during an attack of asthma concentrates his attention on something else he will get some relief. Whenever I feel that I am likely to get an attack of asthma, before getting into bed, instead of inhaling some powder or taking a dose of medicine, I sit up and read a book—either an exciting novel or a scientific work which requires great attention—until, late in the evening, at perhaps 1 a.m., I feel too tired to continue. I then get quickly into bed, fall asleep at once, and sleep through the night without getting an attack. Or if I wake in the night with a severe attack, I sit up and read some similar book, concentrating my entire attention upon it. The dyspnœa is soon forgotten and gradually disappears, so that within an hour it is generally possible to go to sleep again. It needs some strength of mind to concentrate one's attention on a book when one is gasping for breath, but the result well repays the mental struggle.

In this connection it should be remembered that many other forms of treatment depend largely on the mental effect they produce. Nearly every asthmatic is convinced that the powder he uses is of more value than any other, although the composition of all of those in common use is almost identical. It is his firm belief in the potency of his powder as much as the effect of the fumes themselves, which

leads to the relief of an attack. Such methods of treatment as the subcutaneous injection of adrenalin, which has been recently advocated, can only act by the mental effect they produce, and the difficulty in obtaining a certain famous American "cure" is the only reason why many patients are convinced that it is more effective than an exact imitation of it dispensed by an ordinary chemist.

(3) *The exciting cause.*—As the nasal mucous membrane is the most common source from which the afferent impulse which gives rise to the reflex bronchial spasm arises, an attack can often be cut short by rendering it anæsthetic by means of cocaine, or bloodless by means of adrenalin. The two drugs are often used in combination and are best applied in the form of a spray. This method of treatment has the great advantage over the inhalation of fumes in that the drugs used are not irritants. But their prolonged and regular use should not be encouraged; I have known an asthmatic patient develop the cocaine habit and become hopelessly insane from the use of this drug. In a good many cases, particularly in children, great relief can be obtained by treating the alimentary canal. An emetic in a child will often cause an attack to disappear at once, and sometimes a purge or an enema will give relief.

There is one method of treatment, which I think is one of the most valuable as well as the least harmful, although I am quite unable to explain its action. It is to give a mixture of caffeine (gr. v) with phenazine (gr. x) an hour before going to bed. In milder cases a large cup of strong coffee contains sufficient caffeine, or the powder may be taken in coffee. Most normal people would be kept awake by such a large dose of caffeine taken at night, but very few asthmatics are affected in this way, and in my experience there is nothing which diminishes the frequency and the severity of attacks so satisfactorily.

(B) *Treatment in the intervals.*—Treatment in the intervals between attacks is far more important than that of the attacks, as a single attack can do no great damage, but the frequent recurrence of attacks, or the development of the "asthmatic state," is almost certain to be followed by the development of chronic bronchitis and emphysema. It is possible for every patient, at any rate in the early stages before the disease has become complicated by bronchitis, to prevent getting attacks. The liability to attacks diminishes with their frequency, so that if a patient can avoid getting attacks for a sufficient period, he will finally lose his tendency to get them. Thus *the only cure for asthma is not to get it.* Every asthmatic can find some place where he is absolutely well if he searches long enough, and having found that place he should live there. Hospital patients, of course, cannot do that, but when anybody can afford to do it, he should remember that freedom from asthma is worth a great sacrifice, not only for his present comfort, but for his prospects of health in years to come. If a child is forced to live at home in a town where he constantly has asthma, he will grow up more or less a physical wreck, and his life will be a short one. But if he lives permanently away from home, at a school in some place where he never has asthma, he will develop normally, and perhaps finally will be able to come back and live at home with impunity. It is impossible to say beforehand what particular place will suit. The best plan is to stay a week or two at various places until one is found where the patient is perfectly well, and having found that place he should remain there.

Just a year ago I saw a lady, who for ten years had had severe attacks of asthma, and during the last three years she had never been free from it for a day. She was much emaciated, she had severe bronchitis and could hardly walk on account of weakness and dyspnœa. I gave her a list of places to try until she reached one where she was well. She soon found such a place, where she remained for five months absolutely free from asthma. She then returned to her former home, having put on over a stone in weight, and she has only had a slight return of her asthma and bronchitis.

Many asthmatics, especially town-dwellers, are well at home, but asthmatic when they go for a holiday in the country or at the sea-side. As a rule they stay on, hoping that the asthma will leave them. It rarely does, so that they return from their holidays exhausted from want of sleep, and suffering from bronchitis brought on by inhalations used in their attempts to get rid of their asthma. Several years ago I decided never to stay more than one night in a place where I had asthma, if I could possibly avoid it, and consequently, in spite of a considerable amount of travelling, I have rarely had asthma on more than two or three consecutive nights.

Much can be done by moral treatment to prevent the recurrence of attacks. A patient who feels sure that he will have asthma when he visits a certain place will have an attack, but if he goes with a light heart and does not think about it, he may perhaps escape. I never had an attack of asthma in Oxford except before examinations. I therefore acquired the habit of taking medicine for a fortnight beforehand and arming myself with drugs and powders for inhalation for the expected attack, which never failed to appear. When up for my final M.B. I made up my mind to have no attack. I went to Oxford three days before the examination without any drugs or powder, and I did not take a course of medicine beforehand. For the first time in my life I had no asthma before an examination.

There is one drug which is very useful as a sedative for the over-excitable centre, and that is arsenic. Taken for long periods in small doses it undoubtedly diminishes the liability to attacks.

If there is a gross abnormality of the nose it should be treated, but in my experience permanent benefit rarely follows. The routine cauterisation of the nose, whether any abnormality is present or not, has been advocated, but any good results of such treatment must be mainly due to suggestion.

For the bronchial mucous membrane, especially when chronic catarrh is present, potassium iodide in doses of 5 gr., three times a day over a long period, is very beneficial, perhaps because it diminishes the irritability of the mucous membrane and renders its secretion more watery.

It is most important that big meals should be avoided. As most attacks occur during the night, if a patient goes to bed at eleven o'clock he should eat nothing after six or seven, and even then his meal should be as small as possible. Some advise a diet poor in carbohydrates, whilst others recommend a purin-free or vegetarian diet. As a matter of fact moderation is all that is required, quantity and not quality being the important factor. It is important also that the food should be well chewed and eaten slowly. Care should be taken to keep the bowels regular.

In conclusion, I wish to lay stress on the importance of realising that it is not sufficient to treat attacks, but that we should endeavour to prevent their occurrence, as if a patient has no attacks for a sufficient length of time his bronchial centre will lose its abnormal excitability, and his liability to asthma will be gone.

July 4th, 1910.

SYPHILIS.

A DISCUSSION* BY

Mr. Ernest Lane, Mr. McDonagh,
Mr. Alexander Fleming, Mr. C. F. Marshall,
Major French, R.A.M.C., Dr. Pernet,
Mr. H. W. Bayly.

(*Concluded from p.* 192.)

Mr. C. F. Marshall said that the present position of the treatment of syphilis appeared to him to be drifting too much into the hands of experimental chemists. They were asked to abandon the simpler preparations which had stood the test of time in favour of new-fangled and complicated preparations, too often of unstable composition. One new preparation was followed by another with such frequency that it was impossible to test their value. With regard to injections he thought the method more suited to acute than chronic conditions, *i.e.* to conditions where rapid action was required. However, he himself preferred inunction in such cases. Injections, no doubt, were useful in the case of the Army and Navy, but were irksome and too expensive in private, and not suitable for the routine treatment of syphilis. All ordinary cases could be treated by injection. With regard to arylarsonates, he considered them not only unnecessary but absolutely contra-indicated on account of the possibility of blindness. Many cases were reported, and many others were not reported. Syphilis was quite bad enough without adding blindness to it. For some time it appeared that the British soldier enjoyed immunity to the dangers of arylarsonates, but he (Mr. Marshall) believed Major French had cast some doubt on that point. The multiplication of new drugs was possibly due to insufficient knowledge of the use of old ones. Potassium iodide was useful in all stages of syphilis. With regard to excision of chancre the evidence was contradictory. The earliest possible excision often failed, but on the other hand, Neisser's experiments seemed to show that excision was worth trying. In most cases it involved too much mutilation, and to be successful, spirochætes must be localised to the chancre; they might be spread by unskilful excision into the general circulation.

* Before the Surgical Section of the Royal Society of Medicine.

Dr. GEORGE PERNET pointed out that on clinical grounds alone the pill method of treating syphilis had not proved satisfactory. That was being confirmed by the Wassermann test in the laboratory. Whilst admitting that inunctions were valuable, he contended that the best method of dealing with the disease was that of intra-muscular injections, not in a routine manner, but in properly selected cases. In private practice he had found it efficient. Calomel was the sovereign sheet-anchor in serious complications of syphilis, such as optic neuritis, threatening cerebral mischief, and in obstinate tongue lesions and palmar syphilides for instance. These injections were at times painful, but not always. Dr. Pernet had found that calomel 40 cgrm. to the cubic centimetre in 5 and 10 cgrm. doses was sometimes well borne *quá* pain. But Zambeletti's preparation was usually practically painless and more reliable from that point of view. As to grey oil, that was a most valuable preparation, less potent than calomel, but very suitable for the usual run of cases. He employed French preparations of the strength of 40 cgrm. to the cubic centimetre as for calomel. With a specially graduated syringe these preparations were not likely to lead to errors in the dose. The insoluble preparations had their drawbacks, but so had all methods of treatment. In a paper read last year before the American Medical Association in Atlantic City Dr. Pernet had gone into this question, as also into the question of the technique, so there was no necessity to go into details here.* Soluble preparations of mercury were also valuable, but they had to be given much more frequently, in some instances daily as in the case of the benzoate of mercury, but they were often useful to start a treatment and test a patient. All recent experience had demonstrated that mercury was the basis of treatment, for atoxyl and derivatives had failed. It was impossible to go into a number of other matters of considerable importance, so Dr. Pernet concluded by stating that syphilis should be treated for at least four years, vigorously during the first two, then less so for another two. After that the patient should have further courses of mercurial treatment at intervals. Generally, Dr. Pernet agreed with the opener of the discussion, whose conclusions were practically in harmony with those he had arrived at himself, independently and from the point of view of private civilian practice.

Mr. H. W. BAYLY: Anything that I can say on the important subject of syphilis and its treatment must necessarily be from the point of view of the pathologist, and I suppose that all will agree that it is in the pathological rather than the surgical or purely clinical aspects that the greatest advance has been made in the last six years, and it is to the pathologist that the clinician or surgeon now turns for diagnosis in difficult cases.

By the help of the reflecting immersion condenser, commonly called the ultra-microscope, the *Treponema pallidum* can be easily detected in scrapings from the site of the inoculation even before any definite chancre has developed.

Much valuable time is thus gained and the patient is saved the discomfort of the secondary manifestations.

I cannot agree with Mr. Fleming's opinion in regard to the value of the Chinese ink method, as it is frequently very difficult to differentiate the various spirochætes by this method, and the diagnostic value of the movements is lost. Captain Harrison agrees with me that this method is far inferior to dark-ground illumination.

Neisser has shown by means of the Wassermann reaction that the earlier that anti-syphilitic treatment is commenced the more probability there is that early and permanent cure will result, and therefore the ultra-microscope, which in the absence of local antiseptics settles at once whether the commencing pimple or ulcer is the site of a syphilitic inoculation or not, must be considered of the greatest possible value, both from the point of view of surgeon or patient.

By the help of the ultra-microscope the treponema can also be detected in scrapings of a papule or mucous plaque, but it is on the phenomenon of the development of a substance in the serum of syphilitics that has the property of combining with another substance present in organ extracts, and in this process of combination using up complement, that we chiefly depend for the diagnosis of late or latent syphilis.

This complement fixing-test, first worked out by Professor Wassermann, and which carries his name, requires a menagerie of sheep, guinea-pigs, and rabbits, as well as the liver of a syphilitic

* Pernet, "The Intra-muscular Treatment of Syphilis, with Special Reference to the Insoluble Preparations of Mercury: a Critical Review," 'Lancet,' July 24th, 1909.

fœtus, before the original technique can be undertaken.

It has been considered by some devotees that the oracle demands too many sacrifices before she will speak and give her verdict, and numerous workers have tried to simplify the test.

But it seems to me that the most essential of all factors in a comparative test such as this is that all factors except the one to be tested should be constant.

In the technique that I use and advocate I mix complement and syphilitic antigen and normal saline solution in bulk, and measure off 2–5 c.c. of this mixture into each test-tube.

The only variant that I use is the serum to be tested, of which I add 3 c.c. to each tube.

In the same way I mix the hæmolytic antigen and antibody in bulk.

Any difference in the amount of hæmolysis between the tubes must, therefore, be due to the only variant factor, viz. the serum tested.

With Hecht's or Bouer's method, however, or the modification of these methods advocated by Mr. Fleming, the only constants we have are the syphilitic and hæmolytic antigens. The complement and hæmolytic antibody as well as the syphilitic antibody are all variants.

Some human sera contain no hæmolytic antibody to sheep's corpuscles; others contain some, but in variable quantities.

The complement content of human serum also varies greatly. Papers by Mr. Fleming and myself giving our different techniques were published in the 'Lancet' of May 9th, 1909.

I agree with Capt. Harrison that a patient's serum is completely decomplementised by heating at 55° C. for ten minutes, and that greater delicacy of reaction is obtained than if the serum is heated for half an hour. Benefiting by the advice and experience of Captain Harrison, I now invariably use two strengths of complement, and can thus obtain a roughly quantitative measurement of the complement fixing power of the serum. Frequently in this way we can obtain an indication that the patient is getting under the influence of treatment, and some hæmolysis will occur in the tube containing the larger dose of complement, although the smaller dose of complement is completely fixed.

By this means I have investigated the therapeutic value of the different anti-syphilitic remedies, and I published with Mr. Chas. Gibbs in the 'Lancet' of May 7th last a preliminary note of the result obtained in 231 cases, which had received various methods of treatment extending over various lengths of time. This was, I think, the first communication on this subject made in this country.

It is interesting to note that Capt. Harrison also found that pill treatment has a very slow action on the Wassermann reaction compared with intermuscular injection.

My work on this subject has shown that six months' pill treatment has no effect on the Wassermann reaction, and that there is no trace of hæmolysis even with the double dose of complement. On the other hand, 82 per cent. of cases treated by inunction, and 35 per cent. of cases treated by injection, showed some effect of treatment after only three months. As far as I have gone, therefore, my conclusion is that inunction is the best form of administration of mercury, and I gather that Mr. McDonagh's large continental experience leads him also to this opinion.

Calomel injection seems much better than pill treatment, but not so good as inunction as estimated by the Wassermann reaction.

Potassium iodide and the arylarsonates have no effect on the Wassermann reaction as far as three months' treatment is concerned.

If the technique advocated by Mr. Fleming is used, besides having the grave disadvantage of replacing constants with variants, any quantitative measurement is obviously out of the question, as if he depends on the patient's serum for complement and hæmolytic amboceptor, as well as for syphilitic antibody, he obviously cannot increase or diminish one factor independently of the others.

I have tried Mr. Fleming's technique and the technique that I use side by side in 200 cases, and have come to the conclusion that in the long run Mr. Fleming's technique does not save time or trouble, and is not so reliable as Wassermann's original technique as modified by me. I have found partial or complete absence of hæmolytic amboceptor for sheeps' corpuscles in 10 per cent. of human sera, and even if the amount of complement were constant, which it is not, these 10 per cent. would have to be done again by another technique than Mr. Fleming's, and the time spent on the first useless test would have been entirely wasted.

The cases most likely to give fallacious results with Mr. Fleming's technique are those intermediate cases where only a small amount of complement or hæmolytic amboceptor is present.

Most authorities also consider that complement in fluid serum deteriorates quickly, and that it is unsafe to use complement more than twenty-four hours old. Yet another source of error, therefore, is admitted if Fleming's technique is used with sera more than twenty-four hours old. I have therefore ceased using Fleming's technique.

I think it is evident that there is hardly any English pathologist who has had any large experience of the Wassermann reaction with the exception of Fleming who is in favour of any simplified method.

It is in the pathology of syphilis that the recent great advance in our knowledge of this disease has been made, and I think that now the surgeon cannot as in the days of old claim this disease as entirely his.

Why a chronic spirochætosis should be considered surgical at all is baffling to the mere pathologist. And yet not long since I heard my eminent colleague who opened this discussion refer to the pathologist as an "accessory" to the the surgeon.

On the contrary I think that perhaps the day may not be far distant when the surgeon will be looked on as the rather dreaded accessory of the physician-immunisator-pathologist, and that his help will chiefly be sought for surgical interference, where his skill in operative technique will render his help essential. In syphilis it is by the aid of the microscope that we learn whether a suspected sore is syphilitic or not; the pathologist alone can diagnose latent syphilis, and he alone can venture an opinion as to whether the patient is free from the disease. The pathologist can say when treatment should be begun, whether it is effective, whether syphilis is the cause of repeated miscarriages, whether the tumour or rash is syphilitic or not, when the patient may marry, etc., and it is only in clear cases that the surgeon can diagnose this disease unaided by the pathologist. I claim, therefore, that, at any rate in regard to syphilis, the pathologist is equal and more than an accessory to the surgeon. The surgeon, gynæcologist, or dermatologist certainly treats the disease, but nowadays his treatment is guided by the light of pathology, as well as by that indispensable clinical experience which the clinician possesses, and which the pathologist does not.

If the future holds a serum treatment for syphilis, as does not appear impossible, will it be possible then to still rank this disease under the surgical banner?

Why should infection with the *Treponema pallidum* be surgical, while infection with the trypanosome, or filaria be medical? Surely all three are a mixture of the two, and generally far more medical than surgical.

Unless the microscope, the test-tube, or the guinea-pig rather than the knife is in future to be the emblem of the surgeon, it is difficult to imagine that syphilis can remain for very much longer a surgical disease, at any rate to any greater extent than tuberculosis can be considered a surgical disease.

In conclusion, I should like to emphasise my opinion that anti-syphilitic treatment should not be delayed till secondary symptoms have developed, but that a scraping of every doubtful primary sore should be made, and treatment begun at once if the *Treponema pallidum* is found. I consider also that the Wassermann reaction should be used as a guide to treatment, which should be continued intermittently until a series of negative reactions is obtained after all treatment has been left off. I consider that oral treatment is the least satisfactory.

I am entirely in accord with Capt. Harrison's opinion that it is high time that legislative steps should be taken to prevent the spread of syphilis. We live in the days of preventive medicine, and it speaks ill for the courage and dignity of our profession that through fear of the anger of Exeter Hall and faddist politicians, we have allowed these unpractical extremists to dictate to us on questions of public health. Surely a united medical profession could insist on syphilis being ranked as a notifiable disease, and on the segregation of infectious prostitutes.

July 4th, 1910.

"LODAL" is a "Tabloid" issued by Burroughs Wellcome & Co., prepared by the oxidation of laudanosine, and its physiological action resembles that of cotarnine in that it produces contraction in the pregnant and non-pregnant uterus. Clinically it has been found useful in the treatment of uterine hæmorrhage.

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CONTENTS.

PAGE

***A Lecture on Femoral Ruptures and their Management.** By C. B. LOCKWOOD, F.R.C.S. 209

***Some Considerations on Pleurisy.** By W. CECIL BOSANQUET, M.D., F.R.C.P. ... 213

***A Lecture on Lupus erythematosus.** By GEORGE PERNET, M.D. 221

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A LECTURE

ON

FEMORAL RUPTURES AND THEIR MANAGEMENT.

Delivered at St. Bartholomew's Hospital.

By C. B. LOCKWOOD, F.R.C.S.,

Surgeon to the Hospital.

TO-DAY I lecture upon femoral ruptures and their management. But first, femoral ruptures are not always easy to diagnose. Nearly all mistakes in diagnosis are due to insufficient examination. We have examples every day.

For instance, a patient with ilio-psoas abscess was thought by many to have an abdominal tumour. She had been examined lying down. But on standing up there was also a swelling below Poupart's ligament. The abscess was opened.

Here is an example of some of the difficulties which may arise in the diagnosis of femoral hernia. An elderly female had had a severe cough, and whilst coughing said she felt a lump in the right groin. She was examined lying down, but nothing could be seen nor felt. Next she was examined standing up, and yet nothing could be detected. Then the lower limbs were examined, for the veins might be varicose, and the swelling a dilatation of the ampulla of the saphenous vein. Again nothing was found. But she was not told that she was or was not ruptured. If this patient had been in the hospital she would have been allowed to walk about, and been examined from time to time to see if a swelling had appeared. This course could not be pursued, so the patient was told that at that moment no hernial swelling could be detected, but that she might pursue her usual life and be examined from time to time; then if a hernial swelling appeared, its treatment by means of a truss or by operation could be considered.

As a matter of fact this patient had been told by one medical man that she had a rupture, and by another that she had not.

I often wonder why anyone should suppose himself able to arrive at a definite and positive conclusion about complicated conditions after a short examination in the consulting room. If you are in doubt about a case, say that you would like to see it again and have time to think it over, and have an opportunity of examining it on a future occasion. Femoral ruptures are difficult to diagnose because they are often small and the patient stout. It must be very difficult to form an opinion about a small tumour through an inch of fat. Next, femoral ruptures pursue rather a curious anatomical course, which you must clearly understand. After descending through the femoral canal, the rupture presents at the saphenous opening. Naturally you would expect it to pass onwards down the thigh. But it turns upwards over Poupart's ligament and forms a swelling over the inguinal canal. When you remember that the swelling may have a narrow neck which cannot be felt, then you will agree that it can easily be mistaken for an incomplete inguinal hernia. More than once I have seen that mistake made. I remember many years ago seeing Mr. Holden make an incision over a strangulated inguinal hernia. When the sac was exposed he found that he had to deal with a strangulated femoral hernia. Here are notes of operations done upon femoral ruptures. One, which consisted of a large lump of fat with a sac in its centre, had been diagnosed as an inguinal hernia. Another on which I performed the radical cure had also been diagnosed as an inguinal hernia. I think these were excusable mistakes. Of course if it had been a question of treatment by a truss it would have been satisfactory to place an inguinal truss on a femoral rupture. But when it comes to an operation, it need not cause embarrassment if the incision reveals that you have to deal with a femoral instead of an inguinal rupture. The mistake is easily put right by making a small additional cut down the thigh. Another reason why it is difficult to discriminate femoral ruptures is that the landmarks are so hard to feel. The landmark which you rely upon is the spine of the pubes. When the finger is on the spine of the pubes and when the rupture is internal to the finger, the protrusion is a complete inguinal hernia. When the tumour is external to the finger the protrusion is either an incomplete inguinal or a femoral rupture. How are you to distinguish one from the other? An incomplete inguinal rupture is above Poupart's ligament, and the finger passed into the inguinal canal ought to feel its impulse. I am assuming that the canal is dilated. Next, a femoral rupture is also external to the pubic spine, and may be above Poupart's ligament upon the inguinal canal. If reducible, a femoral rupture ought to reduce downwards, backwards, and upwards; the inguinal rupture would slip straight back into the abdomen. To my mind the fallacy of this depends upon feeling the pubic spine. In some stout females the pubic spine cannot be properly felt. If the hernia is reducible it should be put back, and then the beating of the common femoral artery can be felt for. The artery is a guide to the situation of the femoral ring, which lies just inside it and beneath the crural arch. When the hernia is reduced one usually finds a depression at the femoral ring. In the diagnosis of femoral ruptures you may be misled by considerations of sex. For instance, you are taught that inguinal ruptures are common in men and femoral in women. But that is a consideration which need not weigh in your minds, because the question is not what is common in men or what is common in women, but what is the matter with the patient who is in your presence. Also, you may be puzzled by a femoral rupture in a boy. I have thrice operated upon boys for the radical cure of femoral hernia. I suppose they had congenital sacs into which a rupture descended. I do not know whether any of you have thought of investigating the condition of the femoral canal in young adults. They not infrequently have small femoral depressions. A reducible swelling at the saphenous opening is not necessarily a femoral rupture. I operated upon a woman who was wearing a femoral truss on a dilatation of the saphenous vein. You would think that that was an impossible mistake for anybody to commit. But anything is possible to those who do not properly examine their patients. Our first rule is to examine the whole patient; the second to examine the whole of the diseased part, limb, or structure, and compare it with the opposite side; and the third is to examine the part or limb whilst at rest. If he who mistook that dilated

saphenous vein for a rupture had requested the patient to remove her garments and stand up, he would have found marked varix of the whole saphenous vein, and would have had no difficulty in tracing the varicose vein upwards to the swelling in the groin. The saphenous vein at its opening through the cribriform fascia has a dilatation upon it, which is called its ampulla. This dilatation may swell up and simulate a hernia. The deception is very close. The vein empties when the patient lies down, fills when the patient stands up, and has an impulse on coughing—all in common with a rupture. The diagnosis of a difficult case may sometimes be cleared up with a stethoscope. Those with much blood circulating in them have a venous hum: I have heard a marked venous hum in a dilated saphenous ampulla. A psoas abscess is rather like a femoral rupture. It appears almost in the same place. It also may have an impulse in it, and it may become small when the patient lies down, and larger when the patient stands up. But if you follow out the rules in diagnosis it is difficult to make a mistake. Examine the whole patient. In the abdomen you will find the swelling along the course of the psoas muscle, and the vertebral column will be bent. An enlargement of Cloquet's gland, which lies in the femoral canal, may also simulate a femoral rupture. Once I mistook Cloquet's gland for a femoral rupture. The patient had been constipated, but not completely, and had vomited; also she had a tender swelling in the femoral canal. Clearly the swelling ought to have been explored. It is necessary to remember that sometimes in femoral ruptures, owing to the smallness of the femoral ring, only part of the wall of the intestine is strangulated. The symptoms are acute. The patient vomits, but has only partial obstruction. The bowels may act a little, and flatus passes on. There is a small, tender swelling in the femoral canal. I had once before seen the small tender swelling of a partial enterocele mistaken for an inflamed gland. I was therefore afraid lest I, too, might fall into error. It is good to confess mistakes; those who have made many mistakes are more charitable in their judgments than are those who think they are always right.

I believe your text-books tell you that you may mistake an enlarged psoas bursa for a femoral rupture. This must be very rare. I can recollect but once to have seen an enlarged psoas bursa which might have been mistaken for a femoral rupture.

Supposing you have to deal with a reducible femoral hernia, then comes the question, How should you treat it? Many will have radical cure done, but if not, then a truss must be prescribed. Again I advise you to have nothing to do with freak trusses. The best type of truss that you can select and learn to understand is the ordinary spring truss. This is similar to an inguinal, and consists of a spring, which goes two thirds round the pelvis, and has a pad almost the same shape as the inguinal pad, but the plate which supports it is bent downwards to hold the pad upon the femoral canal instead of upon the inguinal. The pad is covered with wash-leather, and is made of a thick layer of felt—the best felt that can be procured, that which is used in the manufacture of pianos—and underneath is a piece of cork. The patient is measured lying down and in exactly the same way as one with an inguinal rupture. A tape is drawn round the pelvis across the base of the sacrum and below the spines of the ilium in the grooves there, and meets at the pubic symphysis. When the patient is stout it is desirable to pull the tape fairly tight. If the patient is thin, the angle of the truss needs extra care. In the last lecture great stress was laid on the angle of the truss. If that be ignored a properly fitting truss cannot be prescribed for a thin person. The angle is below the anterior superior spine of the ilium, where there is a sharp bend at the junction of the thigh with the haunch-bone. This femoral truss only differs from an inguinal truss in the position of the pad. Observe that the pad of an inguinal truss goes straight onwards to rest upon the inguinal canal, but this femoral truss is turned downwards so as to cover the saphenous opening. Sometimes the pad is made a little hollow towards its outer side to prevent compression of the common femoral vein. In prescribing you would say, "Be so good as to supply a thirty-six inch femoral truss. Right side, medium spring, usual shape (or sharp angle), wash-leather cover, ordinary pad."

A femoral truss is put on whilst the patient is lying down. It is slipped over the thin part of the thighs and pulled upwards until it rests on the saphenous opening. Before the truss is put on the rupture should be reduced. You had better

request the patient to reduce the rupture. It may have some peculiarity of which you are not cognisant. A femoral rupture is peculiar in the methods of its reduction: you reverse the way in which it came down. At the beginning of this lecture I said the rupture descended the femoral canal until it had reached the saphenous opening, and then it came forwards and went upwards. You now have to reverse that process. Place the fingers of one hand against the lower part of the swelling. That will prevent you pushing the rupture down the thigh. Next, begin to knead the rupture downwards as if you were pushing it downwards over Poupart's ligament, and when you feel it has got to the level of the saphenous opening push it backwards, and then, with the hand which was steadying it below, push it up the femoral canal and then apply the truss. Take every opportunity of practising these manipulations. I used the word "knead," because sometimes by gently squeezing the rupture you can squeeze some of its contents back into the abdomen, and the rest slips backwards more easily. If the femoral rupture is not controlled by the ordinary type of femoral truss, you may succeed by using one with a band round the thigh. That, to my mind, corresponds to the scrotal truss which is used in the treatment of long-standing inguinal herniæ. If the thigh-strap does not succeed, ask the patient to stand up and cough, and observe where the protrusion takes place. The tendency of the rupture is to go upwards, so that you may succeed by having a prolongation of the truss upwards. But nowadays, if difficulties are met with in the application of a truss, doubtless the patient will elect to have the radical cure done. A great many femoral ruptures are irreducible. You will remember the warning about being sure of the contents of ruptures; this applies very strongly indeed to the contents of femoral sacs. That which gurgles and goes back easily is obviously intestine, but you do not know whether there is not also a strip of omentum.

A patient had an irreducible femoral rupture which had been said to contain the ovary. Ultimately the rupture became strangulated and the "ovary" turned out to be a bit of fat. A clot in the ampulla of the saphenous vein may mislead. I saw my colleague Macready cut down on what he thought was an irreducible femoral hernia and expose the ampulla of the saphenous vein with a large clot in it. And I recollect the same thing happening to myself. It is desirable to have some idea why these femoral ruptures are irreducible, otherwise you may spend a great deal of time, and hurt your patient very much, in your efforts to reduce them. The commonest of irreducible femoral ruptures consists of a large lump of fat with a small sac in its centre. If you have not seen this condition you will be very much puzzled. In my next lecture I shall tell you more about femoral ruptures and operations upon them.

Now as regards the other points in the treatment of irreducible femoral ruptures. A femoral rupture which is not treated may attain very large dimensions. In the year 1904 a patient under my care in the hospital had a neglected femoral rupture which was eighteen inches in circumference and nine inches in height. It was treated in the following way: The end of the bed was lifted eighteen inches and elastic pressure applied with an Esmarch's bandage. After a time most of the protrusion went back and radical cure was performed. At the operation the sac was large and thick, and had in it a piece of adherent omentum After the neck of the sac had been secured, five sutures had to be put through Hay's and Cooper's ligament to close the gap which was left. As a rule one or two sutures are enough to close the femoral canal. There are other causes for the irreducibility of femoral ruptures besides adherent omentum. Femoral sacs have a curious way of becoming cystic. There may be a single cyst or femoral hydrocele, or two or three cysts may be opened before one is found which opens into the abdomen. Do not try to tap femoral hydroceles. Once a house-surgeon tapped a femoral hydrocele and drew off yellow fluid. I afterwards performed radical cure and found a sac full of fluid and communicating with the abdomen. Remember you do not know what you are tapping; the sac may contain adherent intestine. I have seen the urinary bladder opened during the radical cure of femoral hernia, and once I found a portion of urinary bladder on the wall of the sac.

These fatty femoral ruptures and those with cysts, or adherent omentum, or irreducible contents, can only be treated by radical cure. Therefore, in my next lecture I shall deal with operations for the cure or relief of femoral ruptures.

July 11th, 1910.

SOME CONSIDERATIONS ON PLEURISY.

A Lecture delivered at the Hospital for Consumption and Diseases of the Chest.

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It would of course be impossible in the space of an hour's lecture to deal with the whole subject of pleurisy, and I shall therefore ask you to consider this afternoon only a few aspects of the condition which seem to be of interest.

First, then, with regard to its *ætiology*. Inflammation of a serous membrane may be brought about by any infective germ which can gain access to it and establish itself therein; and such access may be gained in several ways. In the first place, entrance may be effected by direct penetrating wounds from without, such as stabs and other wounds through the chest-wall, but in ordinary civil life this is an uncommon cause of pleurisy. Wounds from within may result in such conditions as injury to the lung by a fractured rib, germs then entering from this organ.

Secondly, there is the important group of cases due to spread of inflammation from some focus of disease in a neighbouring organ, and a consideration of the parts with which the pleura is closely related at once indicates the variety of such causes. We have to consider the lung, the chest-wall, the diaphragm, the heart and pericardium, the œsophagus, and the lymphatic apparatus in the mediastinum. Cause arising in the lung will immediately occur to you. In the foremost place we may put acute pneumonia, in which there is always some degree of fibrinous pleurisy over a consolidated area if this reach the surface of the lung, and in which serous effusion and even empyema are not uncommon. With tuberculosis of the lung I will deal separately, but one special accident in this disease which may be mentioned here is perforation of a cavity into the pleural sac, with formation of a pneumothorax or generally a pyo-pneumothorax. Infarcts of the lung generally show a patch of pleural inflammation over them, and the pain which accompanies them is thus explicable, and nodules of malignant disease at the surface of the lung may cause affection of the surrounding pleura. In actinomycosis of the lung the pleura is always densely adherent; and in many cases of bronchiectasis in which the inflammation extends outwards from the ulcerated tubes, the infection may reach the pleura and cause adhesive pleurisy. In many cases of simple bronchitis, if it may be so termed, pleurisy seems to occur, as it very easily might do theoretically, when the terminal bronchioles are infected; but here the possibility of tuberculosis must always be born in mind, or of the presence of a small patch of consolidation (broncho-pneumonia) which may have escaped observation.

Conditions of the chest-wall which may give rise to pleurisy are not very numerous, but a carious rib may cause an abscess which may burst inwardly, and cancer may extend inwards from the breast.

Much more important is extension through the diaphragm, for in this way the thorax may be infected from the abdomen. Suppuration under the diaphragm—subphrenic abscess—is not at all rare, and may arise in perforation of the stomach or duodenum, in pancreatic disease, and in appendicular inflammation, in which it spreads up behind the colon. Bacteria then pass along the lymphatics, traversing the diaphragm, and give rise to trouble in the chest. Usually this is purulent, but it may happen that a serous effusion is found in the pleural cavity, whereas the condition under the diaphragm is purulent. It may occasionally be found that the thoracic symptoms predominate, and an empyema is alone recognised, while the original focus of disease is overlooked; hence it is important to bear in mind the possibility of an abdominal origin for such a condition, and we must return to this subject again under the heading of "Diagnosis."

Spread of inflammation from the pericardium to the pleura is not common, the reverse process being much commoner. The possibility of an infective infarct in malignant endocarditis has already been mentioned. The exact mode of causation of the effusion which occurs in some cases of cardiac disease, generally on the left side, is not clear; but it may be due to extension of inflammation or to pressure of an enlarged heart on some lymphatic channels.

Cancer of the œsophagus may cause infection of the pleura, and owing to the singular latency of this disease, in some instances the pleural signs may be the earliest to be noted, just as in others

invasion of the lung-tissue with symptoms resembling abscess or gangrene of the lung may be the most prominent feature.

Finally, the structures in the mediastinum may be the primary source of trouble. The thymus gland and its remnants must not be forgotten, as tumours arising in it, and abscesses in rare cases, may be accompanied by pleurisy. Tumours, generally lympho-sarcomas starting in the lymphatic glands of this region, may also lead to pleural effusion. In the rare condition—perhaps less rare, however, than is sometimes supposed—chronic indurative mediastinitis, pleural effusion usually occurs at some period of the disease.

Finally, we have to consider cases of pleurisy which appear to be primary. It may at first sight seem difficult to imagine how an infective germ can establish itself primarily in the pleura, situated, as this is, deeply in the body. But it is likely that such a condition may be brought about in two ways: either (1) by a lymphatic route, starting in the neck, since the pleural lymphatics anastomose with those which drain the throat, tonsils, etc., or from the lungs, since there is also an anastomosis between the pulmonary and pleural lymph-spaces, as may be proved by the discovery of particles of pigment which have been inhaled, lying just beneath the pleura; or (2) by way of the general circulation, in which germs may remain virulent for a time and be deposited in some spot of diminished resistance.

In children the pneumococcus may thus attack the pleura primarily or in connection with other serous membranes, two or more of which may suffer at the same time. To the combination the cumbrous name of "polyorrhomenitis" has been applied.

A point of greater interest is the possibility of tubercular infection of the pleura, apart from, or at all events without signs of, coincident pulmonary disease. The view has gained ground in recent years that many, if not all those cases which used to be called "idiopathic" pleurisy, are really due to the tubercle bacillus. Especially likely to be due to this cause are those instances in which a large pleural effusion gradually accumulates without the patient being aware of any disease going on within the chest—such cases as we not very infrequently see in the out-patient department of a hospital, in which a person walks in complaining merely of shortness of breath on exertion, and in which one finds on examination that the whole of one side of the chest is full of fluid. Investigations have been made in various ways to ascertain the relations of pleurisy and tuberculosis. In the first place, the fluid withdrawn may be examined for bacilli; and while it is easy to fail in finding tubercle bacilli in cases undoubtedly due to this organism, we are probably justified in believing that failure to find any other causal organism creates a strong probability that this particular germ is at work. Special methods of investigation have also been devised for facilitating the search for tubercle bacilli, as, for example, the method called "inoscopy," in which the pleural effusion is caused to coagulate, the clot thus formed is digested with pepsine, and the residue obtained by centrifugalising; the resulting fluid is stained for bacilli. A more trustworthy method is injection of a guinea-pig with some of the fluid, using for the purpose a fair quantity, such as 5 or 10 c.c. The tuberculin test has also been applied in cases of pleurisy, and it has been found that between 80 and 90 per cent. of the cases give a positive reaction.

Various observers have endeavoured to trace the after-histories of patients who have suffered from pleurisy, and it has been found that a considerable percentage of them subsequently develop pulmonary tuberculosis. On the other hand, many sufferers from phthisis give a history of previous attacks of pleurisy. Thus Von Ruck, who has collected much information on this subject of the relationship between pleurisy and tuberculosis, found that of 1000 cases of pulmonary disease, 201 had previously had pleurisy, forty of these cases exhibiting it as the initial symptom of the malady. In my own experience the figures are not so large, as I find that out of 1200 cases of phthisis, 5 per cent. had had previous attacks of pleurisy and recovered from them, while another 4 per cent. traced the onset of their trouble to an attack of pleurisy which was followed by general symptoms without any interval of apparent recovery.

I may here, perhaps, allude to an aspect of this relationship between pleurisy and tuberculosis which is not often touched on in the text-books—I mean its bearing on the problem of life insurance. Medical officers of insurance companies may have to decide whether to accept or to decline an applicant who has had pleurisy, and if they accept him, whether he should pay an increased

premium. My own opinion would be that, *primâ facie*, an attack should be looked on as due to tuberculosis, and that therefore it is unsafe to accept such an applicant till some fair interval of time has elapsed, in order that we may see if recovery is complete and permanent. I should entirely decline such a case until two years had gone by. If then all signs had disappeared, while his general physique, his circumstances in life and his family history were all satisfactory, he might be accepted at an increased rate. Possibly when five years had elapsed, if he remained in robust health, he might be taken as a normal life, and I think many offices would so accept him; but in view of the after-history of such cases, as noted just now, I think an additional premium might well be demanded till the applicant was well advanced in life.

Lastly, there is the question of the relation of pleurisy to rheumatism. In past times there seems to have been a tendency to call a variety of affections "rheumatic" when their true causation was unknown, especially if they could be attributed to the action of a chill. We have now attained to rather greater precision in the connotation of the term, but until the specific cause of rheumatic fever has been identified, we cannot distinguish with certainty what different affections may be due to its action. Unfortunately the microbe of acute rheumatism has not been found, or rather no one of several which have been isolated has been satisfactorily convicted as the true criminal. In the case of pleurisy there is an *à priori* probability that the condition may own a rheumatic origin, as such allied structures as the synovial membranes of the joints, the endocardium and the pericardium undoubtedly suffer. And cases of undoubted pleurisy occur from time to time in the course of, or as sequels of, acute rheumatic attacks. One also finds a history of pleurisy in a considerable number of patients who suffer from rheumatism; indeed, in looking through my cases at this hospital for the purpose of this lecture, I have been struck by the frequency of the coincidence of a history of the two diseases. Hence I think we must admit rheumatism as a true cause of pleurisy.

With regard to the *pathology* of the disease I need not say much. I should like, however, to point out that there is no essential difference between the dry or plastic form, and those characterised respectively by serous and by purulent effusion. These are all different degrees of inflammation, dependent upon the virulence of the bacteria at work. Thus the pneumococcus may give rise in one case to a fibrinous or plastic pleurisy, which we call dry, at another to a sero-fibrinous fluid, and at another to a definite empyema with a large quantity of pus. It is also important to remember that in one and the same case an effusion may change its character; and because we have one day drawn off simple serum in exploring the chest with a syringe, it by no means follows that at a subsequent investigation or at the necropsy we may not find pus present.

A second point is that of the conditions under which the fluid in cases of effusion exists. Normally, there is no doubt a continuous process of secretion by the serous membrane, balanced by an opposite process of re-absorption. In inflammation the secretion is increased—corresponding with the œdema of a superficial focus of inflammation—and possibly the absorptive capacity of the pleura is coincidently diminished. Still, it is not abolished; and as recovery takes place the fluid subsides, being drained off by the veins and lymphatics. When, however, there is so much pressure within the pleural cavity that these small vessels are temporarily obliterated, it becomes difficult or impossible for such absorption to occur. Hence the very magnitude of the evil tends to perpetuate it, apart from any further inflammatory action. This is one great reason for artificial evacuation of the fluid in the case of large effusions. Another is the condition of the lung, which collapses in consequence of the pressure to which it is subjected. Like other parts, *e.g.* joints, which remain fixed in one position, it tends to lose its power of re-expansion as time goes on, while the inflammatory process is liable to give rise to fibrous adhesions, which actually bind it down in its collapsed condition, and prevent it from resuming its functions when the fluid disappears.

The sequels of pleurisy, due to the formation of adhesions, must also be borne in mind, and we shall have reason to refer to them again in connection with treatment. The inflammatory process causes destruction of the surface-endothelium of the pleura, and this is replaced in chronic cases by what is practically granulation-tissue; hence arise the adhesions between the lung and the chest-wall

when these granulating surfaces become united, just as the two surfaces of an incised wound do, by scar-tissue. The tendency of such tissue to contract is well known, and by its action traction will be exerted upon the lung and the chest-walls. In these cases there is usually some extension of the inflammation along the connective-tissue septa of the lung, so that some fibrosis of the organ takes place, increasing the amount of scar-tissue present and the degree of traction exerted. If adhesions are formed, then, between the lung and the chest-wall, both behind and before, two results may ensue: retraction of the chest, and pulling open of the bronchial tubes. In the tubes thus dilated secretion tends to stagnate and decompose, and we are then in the presence of the morbid condition known as bronchiectasis. On the other hand, the retraction of the chest may go so far that actual curvature of the spine may result. I need not deal here with the results that may ensue in cases of empyema in which the suppurative process continues, giving rise to septic poisoning with hectic fever, and perhaps amyloid degeneration of internal organs.

With the clinical features of pleurisy in its various forms I propose to deal only from the point of view of *diagnosis*. For this purpose we must consider the dry or plastic form and the type characterised by effusion separately.

In dry pleurisy, in which the striking feature is usually the pain present in the affected side, diagnosis has to be made from other painful conditions, such as intercostal neuralgia and rheumatism, injuries, such as broken ribs and rupture of muscular fibres, inflammatory affections, such as periostitis and perichondritis, and affections of the breast. We have also to endeavour to recognise conditions underlying pleurisy, such as tuberculosis and pneumonia. Some degree of local tenderness may exist in all these cases, but is naturally more marked in such cases as fractured ribs, periostitis and so forth. There is no fever in cases of intercostal neuralgia—and with this we may classify cases of herpes zoster, which may come on with severe pain in the side before the eruption appears or may leave neuralgia as a consequence—and none as a rule in intercostal rheumatism, if that is the right name for cases of pain in the side in rheumatic subjects. The typical signs of pleurisy include, beside pain, rapid shallow breathing and short dry cough; these are absent in the conditions just enumerated. A friction-sound may be audible and will clinch the diagnosis. It is often, however, absent. The distinction between acute pneumonia and pleurisy is often difficult, the mistake of calling pneumonia pleurisy being quite common. The pain at the onset of pneumonia is no doubt pleural, but the more marked rapidity of respiration, the movement of the alæ nasi, and the alteration in the ratio between the rates of the pulse and the respiration may point to the graver disease, in which also the general symptoms are more pronounced (rigor, high fever, headache, perhaps delirium).

The possibility that the pain of pleurisy may be felt over some part of the abdomen—usually in the hypochondrium, but sometimes as low down as the iliac fossa—must also be mentioned, since occasionally it may simulate appendicitis or some such condition. Diagnosis is not difficult, if a careful examination be made, for there will not be any rigidity of the abdominal muscles, as occurs, for example, in appendicitis, while there will be diminished movement of the affected side of the thorax and shallow respiration. Examination with the X rays will show diminished excursion of the diaphragm on the affected side.

When effusion is present different problems in diagnosis arise. In the first place the recognition of small quantities of fluid may not be easy—indeed, it is often impossible without an exploratory puncture. The physical signs presented by a small effusion are the same as those found along with mere thickening of the pleura, such as occurs when an effusion has subsided—dulness on percussion, and weakness or absence of breath-sounds, voice-sounds, and tactile fremitus. Even the X rays do not enable a certain diagnosis to be made in these circumstances. With rather larger effusions some displacement of the heart will probably be discoverable, and will serve to distinguish fluid from mere thickening of the pleura. Two other signs may also be looked for and may assist in the diagnosis, viz. tympanitic resonance on percussion at the apex of the lung on the affected side—the so-called "Skodaic resonance"—and the nasal twang of the voice-sounds as heard at the upper border of the effusion, which may amount to actual ægophony. Absence of these signs does not, however, exclude the existence of fluid in the pleura.

A peculiarity which may lead to an erroneous

diagnosis is the discovery of well-marked bronchial breathing over an effusion. This sign, which suggests consolidation of the lung, is very common in cases of effusions of considerable size occurring in children, and in them exploratory puncture may be desirable for diagnostic purposes in the presence of this sign. Rarely the same peculiarity is met with in adults. In elderly persons the presence of abnormal signs which suggest, but do not quite correspond with, those of an effusion into the pleural cavity, it is well to bear in mind the possibility of a new growth being present in the lung. Collapse of the lower part of the lung may occur in such instances, and be followed by the outpouring of some fluid into the pleura of an œdematous nature to take the place of the shrunken organ. There may then be signs of fluid at the base, and at the same time dulness and perhaps bronchial breathing over the upper part of the chest on the same side. In such cases there is usually a history of progressive loss of strength and difficulty in breathing, and there may be emaciation, and occasionally the presence of blood in the expectoration, both suggestive of tuberculosis. These growths sometimes occur in comparatively young persons, being then lympho-sarcomas originating in the lymphatic glands or thymus. I have also seen a fibro-sarcoma in quite a young woman.

Another question in diagnosis is that of distinguishing between the different varieties of pleural effusion which may occur—between, for example, cases of inflammatory effusion and of passive exudation respectively, and between the different causes to which the effusion may be due. Examination of the fluid withdrawn by aspiration may be directed to ascertaining the quantity of albumen present, and to the recognition of the cells which may be present in the fluid. The examination of the fluid for bacteria has already been alluded to, the most important being the tubercle bacillus, which may be sought either by staining some of the sediment or by inoculating a guinea-pig. A negative result obtained on inoculating a tube of broth or serum with some of the fluid is suggestive of tuberculosis, as the bacillus does not grow readily on ordinary media, whereas most of the pyogenic organisms and the pneumococcus may be grown without much difficulty. In tubercular cases the cells present in the effusion are chiefly lymphocytes—small leucocytes with single nuclei—while in cases due to other bacteria they are mainly polymorphonuclear leucocytes. It seems that lymphocytes occur in effusions due to chronic irritants; polymorphonuclear cells in those which result from acute inflammation. Tuberculosis and syphilis are thus the chief causes of lymphocytosis. In dropsical effusions and in those due to malignant tumours, desquamated cells from the pleural membrane—large epithelioid cells—are often found. In the case of a tumour, however, it may happen that fragments of the growth may break off and small masses of the cells of the tumour may be found floating in the fluid. A lymphocytosis may be present in some of these cases, pointing to the chronic irritation caused by the growth. Hence the last sign cannot be absolutely relied on as an indication of tuberculosis. An effusion containing a large quantity of fibrinous coagulum is suggestive of a pneumococcal origin. In dropsical effusions, on the other hand, the amount of albumen is much less than in inflammatory fluids. For example, in the latter there may be 4 per cent. or more present, whereas in the former there is not usually much over 1 per cent. The albumen may be estimated by diluting the fluid and testing it in Esbach's albuminometer, or by acidulating and boiling it, filtering off the coagulum and weighing it. It is not very often, however, that much difficulty of diagnosis arises in this respect.

Traces of blood are often seen in fluid withdrawn by puncture, arising from wounded vessels along the track of the needle. Such traces of blood are not intimately mixed with the fluid. An effusion which is definitely bloody will probably be due to tuberculosis or to a new growth. In the latter event the fluid will rapidly re-accumulate after it is withdrawn by tapping, and will probably continue to contain blood on every occasion when the operation of aspiration is repeated. A source of error which once came under my notice was the rupture of a thoracic aneurysm into the pleural cavity. The patient was admitted grievously ill and with signs of a large left pleural effusion. Blood-stained fluid was withdrawn by the house-physician on puncture. Death occurred some hours afterwards, and a large aneurysm was found, which had first leaked into the pleura and had finally caused death by hæmorrhage. Rupture into the pleura in these cases is more often almost sudden, and there is no opportunity for

such an error in diagnosis as was made in this instance.

A curious condition occasionally seen is the occurrence of a milky effusion in the pleura. This is less common than in the abdomen, but is due to the same causes. Actual fat may escape into the fluid if the thoracic duct be ruptured or wounded. Fatty degeneration of tumour-cells may also give rise to a small amount of fat in the effusion, but more often the substance which causes the turbidity is not true fat, but either cholesterin or a peculiar form of globulin, both usually arising from some breaking-down tumour. Cholesterin crystals may be recognised under the microscope as the well-known squares with corners cut out. The globulin may be removed by boiling, while it is insoluble in ether, which dissolves fat.

I mentioned above the connection of pleural effusion with suppuration beneath the diaphragm. A careful series of inquiries into the history of the illness will generally elicit an account of past digestive troubles, pointing to abdominal disease, and the presence of the *Bacillus coli* may be indicated by the offensive smell of the effusion.

With regard to *treatment*, we must again consider the different varieties separately. In dry pleurisy the chief treatment needed is in the direction of relief of the severe pain. This sometimes passes off quickly of itself, and it is at any rate well to try simple measures of relief first. Of these, hot applications are the readiest to hand, in the form of ether fomentations or poultices. They must be used really hot, not merely warm, and there is a certain amount of art in the use of a poultice, which is sometimes deficient in domestic nursing. Needless to say, the water should be boiling; the basin used should be warmed with a little of this water before use; the linseed should be quickly and thoroughly stirred into a little of the boiling water placed in the basin, and enough should be added to make a stiff porridge, not a sloppy mess. If the poultice is fairly dry to start with a greater degree of initial heat can be borne by the patient, and the efficacy of the application is thereby increased and prolonged. It should be covered with a layer of waterproof material, and then by a thick layer of cotton wool. The addition of a certain amount of mustard to the poultice, varying the quantity from a tablespoonful to four or five in a moderate-sized poultice, increases its counter-irritant effects. The amount of mustard used must be graduated by the delicacy of the patient's skin. A mustard leaf is also a useful application, and is easy to use.

The use of a blister may also be of service, but is rather more disagreeable in its after effects, and rather adds difficulty to the process of auscultation, as one cannot apply a stethoscope over the sore area.

A still more effectual remedy for pain is the application of leeches, but this is only possible in strong patients who can afford to lose some blood. The leeches are put on over the painful area, and the exact spots selected are usually inmaterial. It is generally better, however, to choose as far as possible points lying over the ribs, as it may rarely happen that the bleeding after the bites is difficult to arrest, and pressure can more readily be applied to the bleeding points when they are over a bone. More often, however, it is advantageous to encourage the flow of blood by subsequent fomentations, as the amount actually swallowed by a leech is not large. The relief of pain obtained by this means is often striking.

In the cases of comparatively slight pleural pain, such as occurs in tuberculosis of the lung, the application of iodine to the chest-wall may be effectual. The preparation usually employed consists of equal parts of the tincture and of the liniment of iodine, the so-called "pigmentum iodi," as the liniment is too strong and the tincture generally too weak for use alone. Some persons with delicate skins find even the pigment too strong, and in them we can only use the tincture. The iodine may be applied daily, or twice a day, till the skin becomes a little red and sore; it should then be omitted for a time.

Apart from counter-irritants, we may endeavour to deal with the pain of dry pleurisy by restricting the movements of the side or by general analgesics. In order to immobilise the side as far as possible, thereby preventing the rubbing together of the inflamed pleural surfaces, we may strap the side with plaster, and for this purpose the use of a single broad strip of strapping, passing round the lower ribs and extending a little beyond the middle line at each end, is as effectual as the more complicated method of applying straps overlapping one another like a surgical bandage. The strip of plaster should be put on while the chest

is in a position of expiration, that is to say, as contracted as possible, so that all excursion is restricted. Sometimes we see that patients instinctively endeavour to do this by lying on the affected side, and thus compressing it; but it may be found that this causes increased pain by adding to the pressure on the inflamed surfaces, without controlling the movements of the ribs, and in such cases the sufferer will lie on his back or on the sound side. When effusion is present, decubitus is almost always on the affected side, so as to leave to the sound lung the freest possible range of movement.

In cases in which we recognise the presence of effusion, the main question is "to tap or not to tap," and the general principle to be borne in mind is that as there is a natural tendency for pathological effusions to be absorbed spontaneously when the cause of their formation subsides, we should not be in a hurry to withdraw fluid until we see that Nature is failing to do it by herself. The first rule, therefore, which is generally laid down is to wait a fortnight or three weeks before tapping, this period to date from the time when we believe the effusion to have formed. We must watch the behaviour of the effusion meanwhile to see if it is increasing, diminishing, or remaining stationary. If it diminishes all is going well, and tapping is not likely to become necessary. If it remains stationary and is only moderate in amount, no harm is taking place, in all probability, while it remains. If it increases, a more difficult situation must be recognised. The increase may be due to a continuance of the inflammatory process at work, and in such cases there is, as a rule, nothing to be gained by interference; the pressure of the fluid tends to hinder the outpouring of more, and if we withdraw it, it will reaccumulate rapidly. Hence we should only interfere if evil symptoms occur. But it is possible that an effusion may increase from mere passive pressure on lymphatic and veins, which may be aggravated by the pressure of the fluid itself—a vicious circle, as it is called—and here it is of course necessary to tap at once. We must be guided by the character of the fluid as revealed by an exploratory puncture, and by the presence or absence of signs of inflammation, such as rise of temperature and leucocytosis in the blood. I need not say that a high degree of leucocytosis would render an exploratory puncture imperative, if one had not been made, as pointing to a probable empyema.

What, then, are the conditions which should lead us to tap a pleural effusion immediately? In the first place, of course, may be put the recognised surgical rule of evacuating pus wherever it exists; but here something more than mere tapping is desirable, and incision and drainage, with or without removal of a portion of a rib, are usually required. It is said that an empyema due to the pneumococcus may be cured by simple aspiration, and in a very debilitated person it might be legitimate to try this method first; but it is of doubtful efficacy in any case, and must be regarded as quite an exceptional form of treatment for this affection.

Secondly, it is advisable to tap one side of the chest whenever there is a bilateral effusion of any considerable size, as it is necessary to have one lung at all events capable of full expansion. We should naturally tap the side which seems to have the most fluid.

Thirdly, the extent of the dulness in the chest is a guide to the amount of fluid present and the degree of pressure which it is exerting, and the old rule used to be to tap whenever the dulness reached the second rib on the front of the chest. This indicates the presence of a very large amount of fluid, and I do not think it is advisable to wait for such an occurrence before tapping. If we see a case for the first time when this point has been reached we certainly should tap at once, but a case in which the upper level of the fluid was considerably lower than this would probably also demand relief.

Lastly, quite apart from any such arbitrary rules as these, which need much judgment in application, we must tap in all cases in which the effusion is producing dyspnœa or serious inconvenience of any kind. We are bound not to let the patient run any risk which can be avoided, and we cannot tell when danger may arise if the heart's action or the breathing become at all embarrassed. Any marked displacement of the heart is in itself an indication for aspiration, apart from urgent symptoms.

I need not describe in detail the operation of aspiration. The use of a trocar and cannula is advisable rather than of a syringe with a sharp-pointed needle, since as the fluid is withdrawn the lung re-expands, and may be injured if it come into

contact with a sharp implement. The vacuum in the aspirator need not be high, and slow withdrawal of the fluid is best, to avoid possible ill-effects. Some authorities recommend the injection of air or nitrogen into the pleural cavity to take the place of the fluid withdrawn, but this is not necessary in ordinary cases. No general rule can be laid down as to the spot to be chosen for aspiration : this must depend on the situation and quantity of the fluid. With large effusions a point near to the angle of the scapula is a convenient one to choose, or one about the mid-axillary line. The patient must on no account be laid on his sound side during the operation, as dangerous symptoms may ensue owing to respiratory embarrassment. Indeed, I saw one case—that of a very debilitated woman suffering from an old empyema—in which immediate death resulted owing to the surgeon's turning her on to the sound side to explore the chest. The withdrawal of the fluid should be stopped if coughing sets in or the patient seems faint. The administration of an ounce of brandy or a dose of ether and ammonia before the operation is a wise procedure.

The only danger of any gravity which may attend the process of aspiration is that of œdema of the lung resulting from the fall in the external pressure upon it. The exact causation of this condition, which is characterised by the expectoration of large quantities of frothy, albuminous fluid, is not very well understood, as it sometimes occurs apart from paracentesis thoracis. It is generally fatal when it occurs.

Finally, we have to remember that our duties are not at an end directly we have safely piloted a patient through an attack of pleurisy. In the first place we have to look upon such a one as having probably suffered from a form of tuberculosis, and to realise that although the acute symptoms have subsided, there are still in all probability latent tubercular foci, which may at any time resume their activity. Consequently we must proceed to put the patient under the same conditions as we should one who was suffering from early or suspected pulmonary tubercle, insisting on a period of complete rest from business, and on his leading an open-air life in the best available climatic conditions, with plentiful food and moderate exercise, under medical supervision. This last should include a record of the evening temperature for some time after convalescence seems established, and regular weighing of the patient at stated intervals, say once a month, as a means of estimating his progress or decline in health.

Even in cases which are not tubercular it is necessary to bear in mind the possible sequels of pleurisy in the form of adhesions, fibrosis, and bronchiectasis. We have to do our best to re-establish as fully as possible the functions of the lung, and to avoid those conditions of shrinking of the chest, and even spinal curvature, which we not infrequently encounter. For this purpose carefully regulated exercises must be used to expand the chest and strengthen the muscles of respiration. The greatest care and judgment are needed in this respect, since it is important not to go too fast and thereby light up again a quiescent focus of disease, while we must not delay unduly lest the mischief be established before we begin to combat it. No definite rules can, of course, be laid down ; each case must be a law to itself. But it is safe to point out that exercise should stop short of fatigue, and that at first a careful watch should be kept on the body-temperature, any rise above normal being a sign to stop the exercise for a time and to resume it again rather less vigorously.

July 11th, 1910.

The Influenza Bacillus in the Respiratory Tract.—Holt holds that the use of the term influenza should be limited to an inflammation or infection excited by Pfeiffer's bacillus. It is of great importance in all infections of the lower respiratory tract, especially during the cold weather. It has very much less to do with acute naso-pharyngeal catarrhs and their complications, otitis, adenitis, etc. It may cause severe general symptoms with few or no local symptoms, even of the respiratory tract; but general blood infection with this organism is very rare. While in most patients the influenza infection causes acute symptoms only, recurrences are exceedingly common, and in some patients the attacks are prolonged, persisting for months. The *B. influenzæ* may be harboured in the secretions of apparently healthy persons for a considerable period, certainly for several weeks; whether such persons may spread the disease is not yet determined. If properly made, cultures are of great value for diagnosis, but the bronchial secretion should be secured.—*Journ. A. M. A.*, vol. liv, No. 25.

A LECTURE

ON

LUPUS ERYTHEMATOSUS.

By GEORGE PERNET, M.D.,
Lecturer on Dermatology, Medical Graduates' College, London.

I WANT to talk to you this afternoon about lupus erythematosus, and in order to make the subject more interesting I have brought a number of coloured drawings and photographs, which will, I think, make the subject more intelligible to you.

In the first place, I consider the name lupus erythematosus is one which might with advantage be given up. When patients with this complaint ask the name of the disease, I do not tell them it is lupus erythematosus, for the reason that the word "lupus" often frightens them a good deal, as they naturally think of lupus vulgaris. I call it erythema centrifugum. Of course patients know that lupus vulgaris is very disfiguring, and at times destructive, the establishment of the Finsen light treatment having done much to give the public information about it. The name ulerythema centrifugum has also been coined for the condition, in imitation to some extent of the older name, *Erythème centrifuge* (Biett). One of the original names for this condition was seborrhœa congestiva, given to it by Hebra.

There are several forms of lupus erythematosus. Patients and medical men sometimes tell me they have heard of lupus erythematosus being cured in a certain way, but the kind of lupus erythematosus must be taken into consideration. The other day a lady wrote to say she had heard they had a serum in Germany for the cure of the condition. That patient had an extensive lupus erythematosus, and I wrote and told her I had never heard of the serum, and that it must be lupus vulgaris which she had heard about, a condition which was quite different. I want you to remember that there are many forms of lupus erythematosus, and because one form has been benefited by ionisation, for instance, was no reason why another should be benefited by the same treatment. You may have to deal with a lupus erythematosus which is circumscribed and discoid, chronic, "*fixe*," as the French call it. At the other end of the scale one meets with acute disseminated forms of lupus erythematosus, which are very often fatal, and which are in some types complicated by albuminuria. There is another form which I have described and called primary acute lupus erythematosus (*Lupus érythémateux aigu d'emblée*).* Nine out of the ten cases I collected were fatal. So at one end you may have a few patches on the face or the scalp, or on both, which is more common, and a few on the extensor surfaces of the fingers, but with very little trouble to the patient, whose health may be fairly good in other respects, and at the other a fatal condition. Moreover, in the circumscribed or discoid cases, after remaining localised for longer or shorter periods, a subacute or acute generalisation may occur. There is also acute lupus erythematosus which is associated with albuminuria, and a very serious condition it is. This form must be distinguished from the one I have called primary acute lupus erythematosus. The difference is one worth insisting on, because the primary form commences by a sudden invasion of the patient's skin. Sometimes this form may be taken at the outset for an acute eczema of the face, for instance. It is only later that the features of lupus erythematosus are discernible. It is extremely rare. But one never knows when one may meet with a case. That is why I am calling your attention to it. Sometimes the condition may present itself as an erysipelatous-looking eruption of the face. I show you the picture of such a case observed by the late Professor Kaposi, of Vienna, and on first looking at such a case you might certainly regard it as an erysipelas. Kaposi called it erysipelas perstans. But it is not really an erysipelas, but part and parcel of an acute form of lupus erythematosus.

In addition to the forms I have mentioned, there is a telangiectatic form of lupus erythematosus, which is not an infiltration or atrophic scarred condition, as is usually the case, with plugging of the follicles and scaling, but there is a telangiectatic process with a butterfly distribution, as in this drawing.

Then we come to a very rare form, which was described by the late Dr. Radcliffe-Crocker, a nodular condition, of which I show you the original coloured drawing.

* Pernet, 'Le Lupus érythémateux aigu d'emblee,' p. 137. Paris: 1908. (A Monograph, with bibliography.)

Here is a plate representing the usual symmetrical lupus erythematosus met with. There are circumscribed areas of inflammatory thickening, with atrophic scarring, or rather atrophy, in the centre of the patches. The nose, cheeks, and ears are usually affected, that is to say, they are the favourite seats. Here you see the hands involved. Whenever you are dealing with lupus erythematosus you should look at the backs of the hands. Here is another form represented which deserves the name given to it by Hebra, namely, "seborrhœa congestiva." It is a seborrhœic-looking condition with surrounding inflammation. Lupus seborrhœicum is another name. This drawing represents another case in which there is a nodular formation. In this next illustration the scalp is affected, with more or less irregular areas with atrophic scarring. But it is not the same scarring as that which follows ulceration, because in lupus erythematosus you do not get ulceration, except very rarely. The morbid process spreads at the margins and clears up at the centre, either more or less spontaneously or else under treatment, and thus you get conditions of atrophy in the areas which have been traversed. That is a very important feature in coming to a conclusion as to the nature of the disease. Here in this next picture we have the same condition of nodulation. Some of these cases simulate other morbid conditions, but that I will speak of later. In the acute cases of lupus erythematosus you get not only inflammatory reaction, but also plasma-cells. In a piece of the skin of the face from my case, already referred to, there were many plasma-cells. In cases where there is an acute onslaught on the system, not only do you get ordinary lupus erythematosus conditions like this, but you may also have bullæ and vesicles and large, raised, erythematous areas. It becomes a polymorphic complaint, which in some cases has led to its being taken for polymorphic erythema. Here is a coloured drawing of lupus erythematosus, from the late Dr. Radcliffe-Crocker's Atlas. Notice in this condition the telangiectatic condition of the cheeks. There are very few cases of this kind about. There are dilated vessels, but on looking closely with the lens you will find signs of atrophy, showing that the process is practically the same. Here, again, is a photograph of a case of my own, in which you will see the whole scalp has been denuded. I have seen several cases of lupus erythematosus in which the scalp alone is attacked. But most frequently you find the scalp and face attacked at the same time, especially the areas behind the ears. In one case I saw, a young lady from the near East, there were very extensive areas of disease about the scalp, and her face was scarcely affected at all. Look at this plate from Kaposi's Atlas, in which the lupus erythematosus is very widespread. I would call your attention to another picture of lupus erythematosus of the face illustrating the crusting and surrounding congestion and inflammatory reaction. Here is another showing extensive invasion of the arms and hands. Also a photograph of a case which I have had under my care, in which the patient had lupus erythematosus about the face and on the front of the chest. She also had a fair number of lesions about the arms and on the back.

Now as to the important matter of diagnosis. One of the things to diagnose this condition from is lupus vulgaris. Although lupus vulgaris begins in a different manner, yet, when it has been going on for many years, one may get a symmetrical condition in lupus vulgaris, superficially similar to that observed in lupus erythematosus *quâ* distribution; but you must of course go into the history, the evolution, the duration, the age of the patient, and so forth.

I show you here a picture of a condition known as lupus pernio, distinguished by the cyanotic appearance of the fingers and hands—very symmetrical, as you will observe. Remember always, however, that there are other diseases besides lupus erythematosus which are symmetrical. Here is pellagra, for instance, about which we have heard a good deal lately. In leprosy, again, we get a butterfly swelling of the face. Dr. Ashburton Thomson, of Sydney, was kind enough to fill in this diagram of a very early case showing the butterfly distribution in leprosy. By the kind permission of Dr. Fordyce, of New York, I am handing round a photograph of a leprous Chinaman, showing how symmetrical the infiltration of the face may be. In diagnosis, therefore, you must not be satisfied with symmetry alone. I insist on this point, for I have seen a diagnosis more than once made on one feature without corroborative evidence. It is important to take a general view first, and that will help you more than immediately

scrutinising a limited area of the skin. I have seen one case of lupus erythematosus in which there were symmetrical patches on the back of almost exact butterfly appearance. That would be an important fact. Lupus vulgaris, from which you must differentiate lupus erythematosus, commences as a rule in one focus, but it may commence in many foci. Especially after measles, children may get five, ten, or more patches of lupus vulgaris scattered about the body. But ordinary lupus vulgaris of the face, which is what you have to differentiate from lupus erythematosus, begins usually in one focus, gradually spreading at the margin and invading more or less the adjacent tissues, ultimately forming a patch ; but as a rule that patch is asymmetrical. It is only when it is very advanced that you get such a symmetrical condition starting from the centre of the face as I show you in this photograph. Lupus erythematosus, on the other hand, has a tendency to develop bilaterally and symmetrically ; it is not limited to one patch as a rule, though it may be, and that is where the difficulty in diagnosis sometimes comes in. Age is of great assistance, because lupus vulgaris as a rule does not begin in middle age but in childhood. So if you get a patch developing at thirty years of age, it is much more likely, especially if it is bilateral and affecting the ears as well, to be lupus erythematosus. After taking a bird's-eye view of the case you examine it more closely. You find in many cases follicular plugging, and if you use a lens, as you should do, you will notice some atrophy or thinning of the tissues, with infiltration of the borders. All these points are in favour of the case being one of lupus erythematosus. But, again, in lupus vulgaris a certain amount of spontaneous recovery may occur. The scarring, however, has a different appearance to the atrophy of lupus erythematosus. It is very rare for lupus vulgaris to attack the scalp separately and apart from a process of continuity spreading from the face. I have only seen one case in which lupus vulgaris occurred separately on the scalp, and that case was one of old standing—lupus vulgaris of the face—and was of the nature of a secondary infection. Again, lupus erythematosus may be taken for late manifestations of syphilis. I have told you that lupus erythematosus as a rule does not ulcerate, but in the case I have in mind there was a little ulceration and crusting about one of the patches if I remember rightly. As the man was a soldier the diagnosis of syphilis had been made without careful examination. But when one looked more closely into the lesions there was follicular plugging and atrophy, which, with distribution, pointed to lupus erythematosus. The condition was distinctly symmetrical. In disseminated cases you may find a number of small lesions scattered about the body, shoulders, arms, thighs, and legs, with a certain amount of adherent yellow scales and a brownish appearance of the parts, especially when glass-pressure is employed. Such a case I have under my care, and it has been labelled psoriasis. In that patient the face and centre of chest present absolutely characteristic lupus erythematosus lesions. Generally speaking, it is much more likely when you get disseminated lesions and patches that the former are part and parcel of the same complaint.

With regard to the ætiology of this condition, we do not know much about the essential cause of lupus erythematosus. The tuberculosis theory is one which is very much insisted on by some, especially by the French school, and there is something to be said in its favour. But even in the very acute cases such as those I have described in my monograph, although some of the evidence supported such a view, yet in some of the cases this was not the case. At any rate in the family history of almost all patients suffering from lupus erythematosus there is evidence of phthisis ; in some patients, again, there are enlarged glands in the neck, which of course may be accidental coincidence, or, on the other hand, be connected with the lupus erythematosus condition. But in many cases of lupus erythematosus of the face there are no enlarged glands, and it is well known how common these glands are in patients who have no lupus erythematosus. In lupus erythematosus patients you often find that they suffer from a chilblain circulation, or actually suffer from chilblains. We know how often chilblains are connected with phthisis. Another thing which is very important is that intestinal toxins no doubt play a great part in the development of lupus erythematosus, and by treating the alimentary tract you will often get a good deal of improvement. Another ætiological factor is exposure to very strong sunlight. In one case under my observation a lady had been in India and exposed

there to very strong sunlight, and she attributed the origin of her complaint to that. I have had other cases in which a similar history was given. But one cannot be absolutely dogmatic about the ætiology in cases of that kind. Still, the fact is that exposure to strong sunlight seems to aggravate the condition very much in some instances, though sometimes mild exposure to sun does good in lupus erythematosus. Cold winds are generally bad for lupus erythematosus and make it worse.

Reverting to the tuberculous theory, some French dermatologists are of opinion, although the tubercle bacillus may not be the essential cause, that the toxins of that bacillus are of ætiological significance. Against this, perhaps, is the fact insisted on by the late Radcliffe-Crocker that when Koch's original tuberculin was injected in a rather haphazard way, lupus erythematosus was not produced. Nor, may we add, is lupus erythematosus common in the phthisical.

With regard to treatment, that, of course, depends on the kind of Lupus erythematosus with which you have to deal. There are many methods of treating these patches. You can use ointment and all sorts of plasters, and they all give results sometimes. But I think a better application in the case of chronic fixed patches, if it is done carefully, is pure carbolic acid. But it must be applied very carefully, and not when the face is to be subsequently exposed to cold, bleak winds, or when the weather is very cold. With regard to X rays, I have come to the conclusion that they do not help us very much in this class of case; sometimes neither harm nor good results, sometimes the case is made worse; rarely is good achieved. Another method of treatment is ionisation, a 2 per cent. solution of zinc sulphate being employed, and that may give good results, but not always. Ionisation does not answer in all forms of lupus erythematosus, even of the chronic type. Another method which was introduced before ionisation is the application of iodine. It is an old remedy, and sometimes it does good, especially when combined with the internal administration of quinine. In the winter-time you can do patients with a chilblainy circulation, or chilblains, good by giving calcium lactate. I have tried it in such cases, and with, I consider, distinct benefit in some instances. In acute and subacute and in disseminated cases there is no doubt that salicin is a very important remedy, and I strongly recommend you to employ it. It is practically harmless, and should be pushed; you can give 15, 20, and even up to 30 gr. three times a day. The late Dr. Radcliffe Crocker introduced salicin in the treatment of skin complaints, and during the many years I was associated with him I never saw trouble ensue from it in lupus erythematosus. Of course, as in all forms of treatment, one should always be on the look-out. There is another drug which is very important, viz. quinine sulphate, and apparently it saved the only case of my series of ten cases* of acute lupus erythematosus d'emblée which recovered. Dr. Payne gave sulphate of quinine many years ago for lupus erythematosus. The sulphate of quinine should be given in the form of an effervescing mixture; in this way patients stand it better. Cases of the disease associated with albuminuria you must treat on their merits. One point I would here insist on, and that is to examine the urine in lupus erythematosus; this should be done at intervals, especially in the generalised disseminated forms.

As to vaccine and serum therapy, the future will show if we can get assistance in that direction. But I may say with regard to the fatal case of acute Lupus erythematosus d'emblée under my care, that polyvalent serum in rectal injection appeared to rally the moribund patient and keep him going for another week.

July 11th, 1910.

We have received a copy of Walker's Medical Loose-Leaf Pocket-Book from Messrs. John Walker & Co., of Farringdon House, Warwick Lane, E.C. This pocket-book possesses an individuality of its own, and its contents may be so arranged as to suit the special requirements of everyone engaged in medical practice, more particularly appealing to the consultant, to the careful student to whom the making of precise and methodical records is an integral part of his work, and to the busy practitioner, for whom time-saving methods, with the additional advantages of practical utility and ready reference, are essential. By an ingenious system of transfer cases this pocket-book forms a permanent record of all cases kept in a convenient manner for future reference.

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* Nine collected cases, in addition to my own.

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CONTENTS.

PAGE

*Vaccine Therapy. A discussion at a meeting of the Hunterian Society by Dr. HALE WHITE, Dr. G. T. WESTERN, Mr. GIRLING BALL, Dr. HORDER, and Dr. BUTLER HARRIS 225

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VACCINE THERAPY.

A discussion at a meeting of the Hunterian Society.

BY

Dr. HALE WHITE. Mr. GIRLING BALL.
Dr. G. T. WESTERN. Dr. HORDER.
Dr BUTLER HARRIS.

DR. W. HALE WHITE said he thought that before a society engaged with practical medicine like the Hunterian, perhaps the best way to approach the subject was to give his experiences in regard to vaccine therapy. During the last two years he had had exactly fifty cases in which vaccines had been given. Of those fifty, forty-five had had the treatment completed, and it had either been successful or had failed. The remaining five were still under treatment. Therefore he thought it would be of interest if he gave his results with regard to the forty-five cases. Of course he did not intend to inflict on the meeting the reading of the notes of the forty-five cases, but he felt sure those present would agree with him it would be fair if he gave samples of the successful and details of all the unsuccessful cases. He had grouped them arbitrarily only as a matter of convenience. Fourteen of the forty-five cases were examples of disease of the genito-urinary apparatus, and of those fourteen, eight were gonorrhœal. As an example of a gonorrhœal case the first was of interest, that of a woman, æt. 49 years, who was married. She had had multiple arthritis for nine months, and was considered by some to have osteo-arthritis. From the clinical characteristics of the arthritis he thought it must be gonorrhœal. No benefit had followed the many lines of treatment which had been tried. The gonococcus was not looked for in any vaginal discharge, as one did not want to excite suspicion as to the nature of the malady. Dr. Eyre found the opsonic index was 4 to the gonorrhœal organism, and she was treated with gonorrhœal vaccine (stock), five millions at intervals of seven days; the

arthritis subsided, and she was cured at the end of a month, having been ill since nine months before. Another case in point illustrating the gonorrhœal trouble he would next relate. It was that of a male, æt. 18 years, who was admitted for severe gonorrhœal acute arthritis. It was one of the worst cases he had ever seen. The wrists were swollen and red, and the left had actually burst and was discharging. Dr. Eyre gave the stock vaccine and injected five to ten millions, injecting seven injections at seven-day intervals. The result was that the arthritis was completely cured. Another example of the good benefit resulting from the use of gonococcus vaccine was that of a woman, æt. 30 years, who was admitted for very severe septicæmia. And he thought every member present, if he had seen her, would have agreed that she would probably die. She had rigors and high temperature, and profound anæmia, and seemed altogether to be in a dying state. She had a profuse purulent vaginal discharge; streptococci were cultivated from that, but it was suspected that the origin was gonorrhœal and that the streptococcal infection had been secondary to that. It was impossible to demonstrate the gonococci in the discharge, because of the large number of streptococci, but the suspicion was confirmed by Dr. Eyre finding that the index to the gonococcus was 3. She had three gonococcus stock vaccine injections, one to two and a half millions, at five-day intervals, and as a result she was completely cured. He was sure anyone who had seen her would have said the treatment saved her life: she was dying when admitted. Another gonorrhœal case was that of a woman, æt. 25 years, whom he saw with her doctor. She was eight months pregnant, and in that case it seemed, from social reasons, almost impossible that it could be that her acute arthritis was gonorrhœal. She had acute arthritis of both knees and ankles and of several fingers, and there was a discharge from the right ankle of pus. Clinically, from the severity of the onset, the rigors, etc., with the high fever, he told the doctor he thought it was pneumococcal arthritis, but as there was a slight vaginal discharge the possibility of it being gonorrhœal was suggested. Dr. Eyre examined the pus from the discharging abscess, which showed numerous gonococci. Autogenous vaccines were employed, one to two and a half millions, and then five millions were used at six-day intervals. She was completely cured, and altogether the benefit was very striking, the improvement commencing as soon as the vaccines were given. In all the gonorrhœal cases the treatment was successful, and in passing he would like to say he did not know any disease for which vaccines were more successful than for gonorrhœal infection. Of the remaining genito-urinary cases six were infections with the *Bacillus coli*. The following was an instance: A woman, æt. 45 years, was seen with Dr. C. Ewart and Dr. Mitchell Bruce. Five weeks before she had pain and swelling in many joints. She had suffered from gout, and now had an acute inflammation of several joints. The ears and the bridge of the nose were swollen, and she had double conjunctivitis and cystitis, with pus in the urine. And the impression of the doctors who saw her was that she was a dying woman, for she was very ill, with a high temperature and great pain. She was vomiting, and the pulse was very rapid. The *Bacillus coli* was isolated from the urine. The opsonic index to that was 0·35. Dr. Eyre prepared an autogenous vaccine. She had a dose of five millions followed by two larger doses. The improvement after the first injection was very striking. She made an uninterrupted recovery. Another striking case was that of a young man who had typhoid fever. When convalescent, but still in bed, he began to get high evening and low morning temperatures, suggestive of the presence of pus, and on March 15th pus appeared in the urine, and he was so tender over the right kidney that he (Dr. Hale White) saw him because the question of exploring the kidney had been raised. The *Bacillus coli communis* and the *Staphylococcus albus* were found in the urine. The index to the *Bacillus coli communis* was 1·45 and to the staphylococcus ·44. He was vaccinated with five million autogenous vaccine of the *coli communis*, and with autogenous vaccine from the *Staphylococcus albus*. He got perfectly well; the temperature began to fall after the first injection. The next case was remarkable on account of the extreme age of the patient. It was that of a woman, æt. 84 years, who was seen with Dr. H. Sharman. She had had hemiplegia, and following on that, cystitis: she had pyrexia with the cystitis. The *Bacillus coli* was isolated from the urine, and a vaccine prepared by Dr. Eyre. She had three doses, and no other treatment was adopted. She would not have the

bladder washed out, and she got well, having made an uninterrupted recovery. He would now mention two failures. One was in a woman, æt. 40 years, who was seen with Dr. H. W. Armstead. She was first seen for a left renal calculus in 1907. This had so destroyed the kidney that it was decided to remove the whole organ. This Mr. Bowlby did. She was much better after that, and then she had recurrent attacks of pain in the other kidney associated with pyrexia, and the urine contained large numbers of the *Bacillus coli*. The index to that bacillus was ·6. She was repeatedly vaccinated, but without improvement, and she refused to entertain the idea of having the kidney explored. Another failure was in a man, æt. 50 years, who came with severe aortic and mitral disease and much retention of urine due to stricture and cystitis. The *Bacillus coli communis* was isolated from the urine and two doses of five millions of bacilli were given by Dr. Eyre, but there was no improvement, and the patient died six days after the last injection, obviously from the severity of his valvular disease.

Of the forty-five cases which he was considering ten might be grouped under affections of the alimentary system, and in seven out of the ten the organism found was coliform. The reason for this conclusion in each of the seven cases was either that the opsonic index deviated from the normal, or an agglutination reaction was given. One of the best cases of the kind was that of a married woman who was sent by Dr. C. S. Pantin. Every three weeks for the last eight months she had had attacks lasting three days, with nausea, heartburn, a feeling of illness and shivering, and a slight rise of temperature. The attacks incapacitated her. During an attack she had constipation. She was out of health and lost much flesh. Duodenal ulcer and atonic dilatation of the stomach had been diagnosed, but she had received no treatment for these conditions. It was found that the washings of the stomach contained large numbers of *Bacillus coli communis*, but no other organisms. From her own *Bacillus coli communis* Dr. Eyre prepared a vaccine, and it was given at intervals of twelve days in doses increasing from five to fifty millions. At the end of six months her weight had increased from 7 st. 9 lb. to 8 st. 10½ lb. She looked so well that she was hardly recognisable as the same person, and her attacks were very slight and infrequent. The washing from the stomach revealed the presence of *Bacillus coli*, but in only very small numbers. She was seen again six months later, and had maintained her weight, had had no further attacks and looked well. That was the most remarkable case in his series, because of the discovery of the *Bacillus coli* in the washings of the stomach. Another good case was that of a boy, æt. 12 years, a patient of Dr. James Smith. He had had ulcerative colitis four years, and was passing blood and mucus, and having pyrexia and abdominal pain and tenderness; no treatment had done any good. Operative interference was suggested, but the parents were opposed to this. The *Bacillus coli* was isolated from the fæces by Dr. Eyre, who prepared a vaccine, and the patient had doses of it, varying from five to fifty millions, at average intervals of a week, for three months. The child was given two pints of lactic acid milk three times a day, and the improvement was immediate and rapid. It was now eighteen months since the treatment, and he was well and was going to school. He saw the boy just before the meeting. Another case was that of a man, a patient of Dr. Shackleton's, æt. 30 years. For five weeks he had severe diarrhœa, with much blood; he was much blanched, and no treatment had done him good. Examination with the sigmoidoscope showed the whole membrane of the rectum to be injected, and the *Bacillus coli communis* was isolated from the fæces. Dr. Eyre prepared a vaccine and various doses were given, and although they had to be given for a long time, the patient recovered under treatment. The next case was also very striking. The patient was seen first two years ago. He had had severe ulcerative colitis for many months. The patient had been confined to bed for a long time, and looked pale and miserable, and had passed much mucus and blood, and the *Bacillus coli* was isolated from the fæces and a vaccine prepared. A small initial dose was given, rapidly increasing to fifty millions, at fortnightly intervals, and the patient gradually improved. The vaccine treatment was continued for ten months. Since thirteen months ago he had not had diarrhœa or the passage of blood or mucus, he had gained weight, and was now hard at work. Another case he mentioned because appendicostomy was done as well as vaccine treatment. A man, æt. 30 years, came into the hospital on April 29th, 1909. He had never been out of England.

On admission he was deadly white, with face sunken, much wasted, temperature subnormal, motions three a day, and consisting almost entirely of bright red blood. Rectal examination showed ullceration within a few inches of the anus, and that was confirmed with the sigmoidoscope, and it was thought he would die, because usually when in an ulcerative colitis the ulceration can be felt with the finger patients do not recover. He was kept in hospital some months; the *Bacillus coli communis* was cultivated from the fæces. He was vaccinated and put upon sour milk and cyllin, and he did very well indeed and gained weight, and was discharged from the hospital apparently cured. His weight increased from 9st. 4lb. to 11st. 2lb. He had altogether nine injections of the vaccine. Dr. Hale White regretted to have to report that they heard he had relapsed after two months, the relapse having apparently been attributable to his having taken a very long walk. He was re-admitted to the hospital and again vaccines were tried, but he did not improve, and so appendicostomy was done; vaccine treatment, both *coli* and streptococcal, was continued since appendicostomy, which was done on February 17th of this year. He had gained 17 lb. in weight and looked quite well, and he hoped that the benefit would continue. Those were all the alimentary cases which he thought it necessary to mention in which the *Bacillus coli* was the offending organism. But he had notes of one in which the offending bacillus was a streptococcus. The patient was æt. 23 years, and came into the hospital under his care. He had never been abroad. Six weeks before admission he had bad abdominal pain and bloody diarrhœa—in fact, he was so ill that he had to go to bed. On admission he was thin and wasted, his temperature was subnormal, the pulse rapid, and he was passing seven or eight watery motions in the twenty-four hours, with much brownish blood and mucus, and very little fæcal matter. The blood-serum did not agglutinate the dysenteric or coli series of micro-organisms. But Dr. Eyre found *Streptococcus fæcalis*, to which the opsonic index was ·54. The patient was accordingly vaccinated with increasing doses of that bacillus, and was also put upon soured milk and cyllin. He left the hospital, having put on 24lb. in weight, and passing no blood in the fæces. Five weeks later he wrote to say he was well and his motions normal, and that he had gained another half stone since he left the hospital. One case was a mixed infection, namely, that of a man, æt. 26 years, who was admitted under Dr. Hale White's care on March 4th. His symptoms were much the same as the others, but the *Bacillus coli* was grown from his fæces and a pathogenic streptococcus. He was vaccinated with both those, and was put on soured milk and cyllin, and he rapidly improved, and after a month his motions were semi-solid and blood-stained, but there was no diarrhœa. A fortnight after this he insisted on discharging himself because he said he was well. But he relapsed and would not come into the hospital again. But he did so well under the treatment that there is very little doubt that if he had stayed in longer he would have received more benefit.

Those cases, he thought, illustrated very well the benefit of the vaccine treatment in cases of ulcerative colitis.

He also had notes of a case which he regretted to say must probably be regarded as a failure. The patient was æt. 34 years, and he had been in India six and a half years, but had never had dysentery. He first noticed diarrhœa in August, 1907, and he passed blood and slime. In 1908 he went to stool twelve times daily. Dr. Eyre was not able to cultivate any pathogenic organism from his fæces, but his opsonic index to his own *Bacillus coli communis* was 1·4, and on the strength of that he was vaccinated, and the usual treatment with sour milk and cyllin was adopted. But the patient did not do well, although the blood and slime got less in quantity, and when he left the hospital he was only passing one normal motion in twenty-four hours, and he had not passed any blood for five weeks. He regretted to say that the patient wrote to him three months later saying he had diarrhœa again, and although he was greatly improved he did not regard himself as cured. It was to be noted in this case that there probably was not success in obtaining the organism which was the cause of the trouble.

The next group of cases which he would like to bring forward were cases of empyemata and subdiaphragmatic abscess. A very good instance of that was a case which he saw with Dr. Brock and Dr. Holman. The patient arrived at Tilbury Docks a mere skeleton, 5½ st. in weight, coughing up over a pint in twenty-four hours of anchovy pus, like expectoration from a hepatic abscess. He was asked to see the patient to see whether the abscess should be drained. On talking the matter over it was decided that the man was too ill to do anything, and by the time he got to his own home he seemed to be dying. Gradually he improved, and the expectoration and pain got very much less; he was putting on weight, and was sent to Cornwall for

open-air treatment. Six months after he landed in England he was unrecognisable. He still had expectoration to the amount of four ounces of pus a day. Dr. Eyre found his pus contained streptococci, chiefly the *pyogenes longus*. He had several doses of vaccine, and it was very striking to see the improvement. As soon as the vaccine was started the expectoration ceased, and a year after his arrival in England we could not find that there was anything the matter with him. His weight was 12 st., and he could walk uphill with ease. The X-ray picture taken by Dr. Jordan showed no evidence that the lung had been discharging large quantities of pus, so complete was the cure. It was computed that he must have coughed up fifteen gallons of pus through his right lung since his arrival in England. He (Dr. Hale White) had heard of the man just before the meeting, and he was still well and following his employment. The next case was interesting because he was a bad subject. He saw, with Dr. Prentice, an old man with melancholia, who had an empyema at the base of his right lung, from which he was coughing up pus. The *Streptococcus lanceolatus* was cultivated by Dr. Eyre, and the vaccine from it was given to the man, who had three injections in six weeks. He improved very much, and a year after the treatment he was well; the expectoration had ceased a month after the treatment was begun. Another which he would mention was interesting, because the patient was a doctor, æt. 60 years, who had duodenal ulcer, and had been losing very much blood, so much that it was decided to short-circuit him. That operation was done by Mr. Arbuthnot Lane on December 26th, 1908. Immediately after the operation the patient vomited blood from the ulcer and sucked some of the blood into his lungs, and got pneumonia at the base of the left lung, which led to an abscess there. During the whole of January his temperature remained high, and he continued to cough up the pus. On January 29th *Staphylococcus aureus* and *Streptococcus longus* were found in it, and a vaccine made from both of them was injected. The temperature then fell at once, and he got well. It was now a year since he went back to his work, and he told Dr. Hale White just before the meeting that he was perfectly well.

A good case of simple empyema treated in the same way was that of a woman, æt. 34 years, who came in on account of expectoration of pus. Physical signs and examination by means of X-rays showed an empyema in the upper part of the right chest. *Bacillus coli*, streptococcus, and the pneumococcus were isolated from the pus, but the *Bacillus coli* was the only one which suggested itself as the cause of the trouble because of the result of the examination of the opsonic indices for the organisms. She had four doses of the vaccine, and under this treatment the expectoration ceased, she gained two stone in weight, and improved in every way. Another X-ray picture taken by Dr. Jordan before her discharge showed great shrinkage in the cavity of the empyema. The rapidity of improvement left no doubt as to the benefit of the vaccination treatment.

Another case of which he had the notes was that of a man, æt. 40 years, who was sent into Guy's Hospital by Dr. Aston Key. Many years before he had empyema, which was operated upon. But the sinus had closed sooner than was good for the patient, for after its closure he began to cough up pus, and he had been doing it for six years when Dr. Hale White saw him. After many failures to find the pus by means of a needle, Dr. Fawcett was fortunate enough to hit it off and the abscess cavity was drained. He was vaccinated, and improved very much. It was difficult to say how much of the improvement was due to the vaccine, but everyone would bear him out as to the difficulty with which a long-standing empyema usually recovered, especially in adults, and in this case it was almost impossible to resist the conclusion that the pneumococcic vaccine which he had did benefit him. He had had several doses, gradually increasing from 5 to 50 millions. Four months after operation the sinus had closed and he was well. It was a very remarkable case, because the man could drain his empyema by hanging his head down and leaning it to one side; in that way he could, in a few minutes, get rid of a pint of foul pus. He (Dr. Hale White) had promised to give an account of all the failures, and therefore he would mention the following: the patient was a man who had been operated upon for empyema, and the sinus had been allowed to close earlier than it should have done. He had a high temperature, and Dr. Jordan examined him with the X-rays and the picture indicated an empyema close up to the spine. That abscess was drained by Sir Alfred Fripp; it was only a small one. The offending micro-organisms in this case were the *Staphylococcus aureus* and the *Bacillus coli*. Dr. Eyre prepared vaccines from these. Many injections were given, and the man had been away to the seaside. But the small empyema could not be got to heal up, and he feared the patient was no better for the treatment. Another failure in connection with a chest condition was that of a man æt. 16 years, who came into Guy's Hospital on account of bronchiectasis, with much expectoration. The *Micrococcus catarrhalis* was the only organism found in the sputum. He had seven injections of an autogenous vaccine, gradually increased from 5 to 50 millions. There was no improvement.

The remaining cases were miscellaneous, and he did not think it was necessary to relate cases of boils and pustular acne. But of other cases there were interesting ones which he might relate. There were three cases of puerperal septicæmia, in which

it was difficult to believe that the vaccine had not contributed to save the patient. One was that of a married woman, æt. 27 years, who was confined on April 11th. He first saw her on April 25th, with general septicæmia and septic pneumonia. Cultivations from the vaginal discharge and from the sputum showed the *Bacillus coli communis* and the pneumococcus, and she was vaccinated with the pneumococcus as the opsonic index indicated that that was the most likely to be the offending organism. The improvement was immediate and remarkable, for when first seen she was thought to be dying, and he doubted very much whether she would not die before a vaccine could be got for her. There was another woman, æt. 30 years, who was confined on June 22nd, and her temperature was 104° F. on the 25th. She had been having frequent rigors until Dr. Hale White saw her on June 23rd. She was then found to have an acute nephritis, with bloody urine, and urine drawn off with the catheter revealed the presence of *Staphylococcus aureus*. A vaccine of this was given, but the clinical conditions remained unaltered; then *Bacillus coli* was isolated from the vaginal discharge, and a vaccine of that was prepared. Only four injections were given, and by July 12th, ten days after vaccine treatment with *Bacillus coli* was commenced, the temperature was normal, and the patient made an uninterrupted recovery. A year afterwards she was seen and found to be in perfect health. The next case he had to relate was a very severe one, that of a woman, æt. 20 years, who was confined on December 7th, and had a rigor on the 9th. On the following day the uterus was cleared out under an anæsthetic. Next day streptococci were found in the blood, and accordingly a stock anti-streptococcal serum was given, but it was useless, and so a vaccine was prepared and injections were given by Dr. Eyre at intervals during the next eleven days. She improved considerably. Under the treatment the streptococci disappeared from the blood, and she seemed to be going on well, but three days later the temperature again rose, there were definite signs of septic pneumonia, and she died. One of the most remarkable cases he had to mention was the wife of a doctor, æt. 30 years. She was taken ill on February 24th with arthritis in the right shoulder, and had a temperature of 105·6° F. Four days later she had swelling of the left foot, with a pink flush. On March 6th blood was taken from the basilic vein and was sterile. On March 8th there was arthritis of the left wrist and elbow, and the whole arm was swollen. An incision led to the exudation of much serous fluid. On March 17th, from further incisions, a pure culture of the *Streptococcus pyogenes* was found. The temperature for the whole month had been very high, usually 104° or 105° F., with rigors and sweats. The first dose of vaccine was twenty millions, given on March 25th. On March 27th the temperature was 104·8° F. in the evening. Next morning it dropped to normal, and it never again reached 100° F. That was a year ago, and the woman was now quite well. Her husband wrote to him to say that the immediate good result after using a special vaccine ought to convert the most hardened sceptic. There was only one now under treatment which he wished to mention, and that was a very intractable case of what would be called by most, rheumatoid arthritis. From time to time the *Bacillus coli* had been found in the patient's urine, and Dr. Eyre had been preparing vaccines for about a year and giving them at intervals, and there was no doubt that the patient's fingers and joints generally were becoming more supple. He heard of her that afternoon, and she was strongly of opinion that the vaccines had done her more good than had any other of the various forms of treatment which had been tried, including visits to spas, before the vaccine treatment.

He thought that at a practical meeting like the present one it would be better to give every failure and some samples of the successes from the treatment. Time did not permit of any generalisations, but in conclusion he might add (1) that he had never known harm follow in use of vaccines if small doses were employed; (2) in acute cases the benefit, if any, was seen quickly; (3) in chronic cases it was more slowly manifest; (4) autogenous vaccines were better than stock vaccines; (5) treatment by vaccines was not intended to replace, but only to supplement, other treatment.

Dr. G. T. WESTERN, from the Inoculation Department of the London Hospital, spoke on inoculation treatment in puerperal septicæmia. He said: I have during the last two years investigated and treated some twenty cases of puerperal sepsis, and in bringing before you some results to-night I hope to hear some discussion on various points in connection with this condition, about which there appears to be considerable difference of opinion.

The fact that authorities differ in their estimates of the mortality in puerperal sepsis from 4 to 50 per cent. indicates that there is a very wide margin within which selection of cases occurs. This selection of cases is, I take it, due to the fact that puerperal sepsis, in its widest meaning, includes a large number of cases where there is a slight absorption of bacterial poisons from a wound in the genital tract, which causes a temporary pyrexia from which recovery is rapid and complete. It is, however, only the really severe cases of sepsis which ever come under treatment by vaccine therapy, so that I do not propose to discuss these milder cases.

It appears that there is considerable difference of opinion as to what is puerperal septicæmia and what is puerperal sapræmia. I find some physicians

do not admit septicæmia unless bacteria are found in the blood-stream.

Clinically a diagnosis of septicæmia rests largely on the temperature, the pulse, or a combination of temperature and pulse.

Bacteriologically a diagnosis of septicæmia may be made on a positive blood-culture. If the result is negative, however, it does not follow that the case is not a septicæmia. It is a well-known fact that a blood-culture may be done on the same case on several occasions and be positive one time and negative another. Further, if several tubes of media be inoculated at the same time some may show growth while others remain sterile. Again, while a blood-culture done in the ordinary routine way may give a negative result, special precautions with regard to medium and quantity of blood taken may give a positive result. This is well shown in blood-cultures in acute pneumonia and typhoid.

The fact is that a septicæmia is seldom, except within a short time of death, a condition in which multiplication of bacteria take place to any great extent in the blood-vessels, but rather a condition in which there is a local bacterial infection around which the tissues have not reacted to form a localising barrier, and consequently bacteria are more or less continually being passed into the general circulation. Here they are probably killed off before long, unless they happen to form an embolus or alight in a thrombus, in which case a pyæmic abscess may occur.

It appears to me, therefore, that apart from sapræmia we have to deal with two conditions not always easily separable. First, a localised bacterial infection in the genital tract associated with more or less toxæmia, and secondly, a true septicæmia having as its origin an imperfectly localised bacterial focus in the genital tract.

Further, I would suggest that the former group includes a large number of those cases which are commonly termed "sapræmia," but which in reality are due to pathogenic micro-organisms.

Various workers have attempted to distinguish between the pathogenic and non-pathogenic streptococci by various means, *e.g.* length of chain, type of growth, staining, agglutination fermentation reactions, hæmolysis, etc. None of these methods, however, appear to be entirely satisfactory.

It is well established that streptococci can almost invariably be found in the uterine discharge from cases of so-called sapræmia and often in pure culture, and I would suggest that many of these cases are in reality well localised infections with a pathogenic micro-organism.

The bacteriology of the genital tract during the normal puerperium has been investigated by various workers with some very divergent results.

The vulva commonly contains streptococci, staphylococci, and coliform bacilli.

The vagina according to some workers contains few bacteria besides the bacillus of Döderlein, Krönig, on the other hand, finds that after the second day many bacteria are present.

The uterus according to Döderlein is sterile in 80 per cent. of cases, 7 per cent. contain streptococci, while in the remainder anaërobic saprophytes are present. Stolz, on the other hand, finds organisms present in the uterus in 80 per cent. of normal cases on the fourth day.

Nicholson and Little failed to find streptococci in the uterus in any case except in association with symptoms of infection. They consider the positive results obtained by other workers to be due to faulty technique.

It must be admitted that in taking cultures from the uterus there is considerable risk, if adequate precautions are not observed, of obtaining contaminations from the vagina; it seems propable, therefore, that the uterus is really, in a large majority if not in all normal cases, sterile during the puerperium.

On the other hand it is generally accepted that in a very large majority of cases of puerperal sepsis the uterine discharge contains streptococci. Whether these streptococci are the primary cause of the disease or only a secondary invasion is disputed. It seems probable, however, that they are the real cause of trouble in the majority of cases.

In treating a case of puerperal septicæmia it is essential that as little time should be lost as possible. It appears to me, therefore, that blood-culture is often not the best method by which to obtain the infecting organism. First, because the result is in an appreciable number of cases negative; and secondly, because a vaccine can seldom be prepared in less than forty-eight hours, and often longer still, by which time in really severe cases the patient may be dead. If, however, a pure culture can be obtained from the uterus, it is generally possible to have a vaccine ready in less than twenty-four hours.

The method by which I take my cultures is that used by Krönig and Menge. The patient is anæsthetised, probably for the purpose of removing any retained membranes or placenta; if not, cultures may be taken without an anæsthetic, but the risk of contamination is greater. After thoroughly douching the vagina the cervix is drawn down so as to straighten any flexion of the uterus on the cervix. The Menge's tube is then passed up to the fundus, and withdrawn again one inch so as to avoid wounding the fundus with the pipette. The rubber diaphragm is then punctured with a sterile Pasteur pipette and material collected for culture. I have only on one occasion failed to find streptococci by this means, and on only three occasions have I obtained a few contaminating colonies of coliform bacilli or *Staphylococcus albus*. The one negative case was a very mild one in which the temperature fell to normal in twenty-four hours. I would sug-

gest that it would be sound practice to obtain a specimen of uterine contents by this aseptic method in every case where retained secundines have to be removed and where there is a question of sepsis, as if this is not done and the case does not progress well, a valuable opportunity of gaining time and material has been lost. I find that physicians are averse to allowing a Menge's tube to be passed except at this operation. A blood-culture may be made at the same time as a matter of interest.

I do not wish to draw any far-reaching conclusions from the cases I have treated, as I am aware that this would not be justifiable with so small a number.

A few mostly isolated cases have been published by different workers, but usually with such meagre details that it is difficult to draw conclusions from them as a group.

Of the twenty cases I have investigated a blood-culture has been made in eighteen cases. In eight of these the result has been positive, streptococci growing in pure culture in each case. Of the remaining ten cases two developed pyæmic abscesses from which streptococcus was isolated in pure culture, three had pyæmic abscesses post mortem, while in five there was no positive evidence of bacteria in the blood-stream. We may therefore assume that thirteen of these eighteen had bacteria in the blood-stream at some period of their illness.

With regard to treatment, three died before a vaccine could be prepared. Five showed such improvement after the uterus had been cleared out that vaccines, although ready, were withheld.

Of the remaining twelve cases, seven lived, while five died. Of the five deaths one occurred forty-eight hours after treatment commenced, one occurred one week after treatment had been discontinued at the request of the physician, while three died in spite of continued treatment.

Of the seven cases which lived four had streptococci in the blood, while the other three, beyond temperature, pulse, and rigors, showed no absolute evidence of bacteria in the blood-stream.

Brief notes of some of these cases may be of interest.

Case 1.—A multipara, æt. 36 years, was confined on April 9th. Normal labour, temperature slightly irregular (98°–100° F.) until the seventh day, when it rose to 105° F., pulse 136. The uterus was explored and some foul membranes removed. A pure culture of streptococcus was obtained from the uterine contents and also by blood-culture. When inoculations with a streptococcus vaccine were commenced on the sixteenth day the temperature was 104° F. and she was said to have signs of pericarditis and pleurisy. The temperature fell to normal in eleven days and she made a complete recovery.

Case 3.—A primipara, æt. 27 years, was confined on November 18th; membranes were partly left. On the third day the temperature was up to 104° F. and on the fifth day 105° F. The membranes came away in the douche and were very foul. A pyæmic wrist-joint was opened on the tenth day. Anti-streptococcal serum was given without benefit. Pyæmic abscesses also formed in the back and calf. All these abscesses contained streptococcus in pure culture. The opsonic index to this streptococcus was ·6 before inoculations were commenced. It rose rapidly to normal and there was a corresponding fall of temperature to 98° F. The temperature then varied for two weeks between 98° and 100° F., after which it remained normal.

Case 4.—A multipara, æt. 28 years, was confined on September 23rd. She had a rigor on the seventh day, followed by repeated rigors for three weeks, when she was admitted to hospital. During the next fifteen days sixteen rigors occurred. An embolus of the left retinal artery occurred on the twenty-first day, and a pyæmic knee-joint and an abscess in the buttock. From the knee-joint and abscess in buttock streptococcus was isolated in pure culture. A vaccine was made from this and inoculations commenced. No more rigors occurred, and the temperature came down gradually to normal. A parametric abscess later pointed and was opened and a pure culture of streptococcus isolated from the pus. This healed rapidly.

Case 5.—A multipara, æt. 37 years, was confined on January 14th. Considerable hæmorrhage. Placenta had to be removed by the doctor. On the third day she had a rigor and was admitted to hospital with a temperature of 104° F. A blood culture was done, but proved negative. Streptococcus was grown from the uterine contents and a vaccine prepared. Five injections were given in fourteen days, after which time the temperature had fallen to normal.

In conclusion I wish to express my thanks to Dr. Russell Andrews and some other members of the staff of the London Hospital for permission to make use of cases that I have treated while under their care.

Mr. Girling Ball spoke of vaccines in cases of acute streptococcus infection. He said: The use of vaccines in the treatment of acute infective processes has, up to the present time, not met with a very enthusiastic reception when the question of the use of these agents has come up for discussion. Let me therefore give you a short *resumé* of some work which it has been my lot to carry out in cases of this type, the micro-organism of infection being the streptococcus. It is now some two years since this particular variety of case first interested me from the point of view of the use of vaccines in treatment. The suggestion that these agents might be of service came about quite accidentally.

I was asked to see a case of facial erysipelas, the constitutional symptoms associated with the local lesion being very severe in character. The general appearance of the patient was one of an acute illness; there had been a high fever for four days, and the pulse-rate was markedly increased. The local condition on the face presented the characteristic signs of the disease which commonly goes by the name of erysipelas. The usual treatment had been adopted, antiseptic applications had been applied. Despite all efforts the patient showed no signs of improvement. Under these circumstances the use of a vaccine was suggested. It was not thought at the time that this would have any beneficial effect, but at the same time no harmful results were anticipated. In the first instance, difficulty arose in arriving at some conclusion as to the size of the dose of vaccine that should be inoculated; on theoretical grounds, however, the introduction of a small number of dead micro-organisms commended itself. Having failed to isolate a streptococcus (the causal agent of this type of disease) from bullæ, which were present on the surface of the lesion, and cultivations from blood obtained by scarification of the skin at the spreading edge of the inflammatory process having failed to give this positive evidence of the infection, it was decided to use a vaccine prepared fron a virulent culture of the *Streptococcus pyogenes*, which I happened to have by me. A five million dose of the dead micro-organisms was injected into the subcutaneous tissues. To my surprise the temperature straightway fell to normal within the next twenty-four hours, and there it remained; synchronously with this the rash stopped spreading, and at the end of a further twenty-four hours had quite disappeared. So accurately did this change coincide with the introduction of the vaccine that it appeared to me that it must have been due to this agent, but at the same time it seemed quite possible that it was merely a coincidence. It was due to the recovery of this patient in this remarkable manner that led me to investigate further into the action of vaccines in acute streptococcal infections.

The list of cases that have been treated in this way may be classified in the following manner:

(1) Those in which the micro-organisms causing the lesions remained localised to the initial site of infection, with the production of toxæmic symptoms.

(2) Those in which micro-organisms, besides being present in some local focus, were also demonstrated in the circulating blood-stream during life.

(3) Those in which the clinical signs suggested that the conditions mentioned in (2) were present, but in which no cocci could be demonstrated.

(*a*) The patients got better, and thus no proof that the cocci were in the blood-stream could be made.

(*b*) The patients died. In some of these cases no post-mortem was allowed. In others the cocci were demonstrated at the autopsy.

In the first place, let me mention other cases of streptococcal infection of the skin, as these stand in a class apart. A selection of cases was made, those in which the lesions were severe and associated with marked constitutional disturbance being chosen. All cases of erysipelas do not call for vaccino-therapy, as the ordinary methods adopted will be quite sufficient to deal with the infection. These remarks therefore only apply to that type of case which has been indicated. Of these, eight cases have been treated in this way. The initial lesion was situated in the head region, and in most cases had its origin in the face. In three cases local foci of suppuration preceded the onset of the more diffuse inflammatory process; in the remaining five cases there was no obvious abrasion or history of such to explain the infection. A *Streptococcus pyogenes* was isolated in pure culture from the suppurating foci in each instance that such was present; from these vaccines were prepared. In the remaining cases attempts were made to isolate the micro-organism from bullæ, and from scarification of the skin at the spreading edge, but resulted in failure. The vaccine under these circumstances had to be made from a stock culture of a virulent streptococcus. Blood-cultures were also made without any positive results.

In age the patients varied from six weeks to forty-five years.

The lesions were strictly localised to the face area in four cases; in three the inflammation had spread to the scalp and neck regions respectively, and in the remaining case, that of a small child, the lesion was of a wandering character, spreading from the scalp to the feet by a continuous process of infection.

The stage of the disease at which the introduction of the vaccines was commenced varied very considerably, the earliest case treated being seen some four to six hours after the onset of the symptoms, and the longest period after infection had occurred, before the first dose of vaccine was given, was eight days.

In three cases there had been recurrences of the lesions before vaccine therapy was adopted; in one of them five distinct exacerbations were noted. Up to the time of institution of this method of treatment the usual remedies had been tried, but synchronously with the injection of the first dose of dead cocci these were omitted. This rule was followed in five of the cases. In the remaining three, vaccines alone were used from the time when the patients presented themselves at the hospital.

Now let me turn to the actual dosage that was used, the methods used as a guide to the treatment, and indications as to the phenomena which were observed.

The dosage was purposely varied, in some instances small doses of the dead micro-organisms being given at frequent intervals, and in others large doses with longer intermissions. The actual numbers varied from two to fifty millions. The vaccines were introduced into the subcutaneous tissues at a site removed from the infected area.

As guides to the treatment, the course of the temperature, the changes occurring in the lesion, and the improvement or otherwise in the general condition of the patient were noted with great care.

It would be unwise to make any dogmatic statements as to the positive value of these agents in such a limited number of cases, but perhaps a few statements as to the various changes noted, and these taken in conjunction with similar phenomena occurring in the cases which will be mentioned later on, will be some justification for the use of these agents under circumstances such as these. It must in the first place be remembered that the ordinary methods had already failed to bring about the desired result in five of the cases, and that these were omitted after the institution of the vaccino-therapeutic methods. Also it must not be forgotten that the cases were selected from the severest types of the disease, and in two cases at least so ill were the patients that it was thought likely that under ordinary circumstances a fatal issue must have been the result. On the other hand, sight must not be lost of the fact that cases of acute streptococcus infection are prone to resolve themselves in a very remarkable manner even without the intervention of any special methods of treatment, and in no type of lesion is this more marked than in erysipelas. At the same time, the vaccines were purposely introduced at different stages of the infection, so that the changes which I will now mention cannot be said to be solely due to coincidence. In five cases the temperature fell to normal synchronously with the injection of the vaccine. In one case a recurrence of the inflammatory process made its appearance after the vaccine treatment had been stopped for a little over a month. The further dose of dead micro-organisms was given with a similar result to that which had occurred on the previous occasion. In the remaining cases a change in the course of the temperature was noted, which, although not so sudden as that already mentioned, subsequently returned to the normal. In two of these the lesion existed a considerable length of time. In the other case the result of the inoculations was very disappointing, as the symptoms had been present for only a few hours.

Another point was noted. Within the next twenty-four hours after the injection of the vaccine, a rise in the temperature occurred which appeared to depend on the size of the dose of vaccine, being slight when small doses were given and increased with the larger inoculations.

The rash as a rule disappeared with the fall in the temperature, and the general condition of the patient rapidly improved.

In those cases which had previously had recurrences no further exacerbations occurred; in one of the cases which had been treated in the first attack, a return of the lesion was seen at the end of a month after the vaccine treatment had been stopped; the rash, however, rapidly subsided after further inoculations and did not again recur.

The remarks already made relate to a specialised form of streptococcal infection. Let me now turn to the type of infection in which much severer lesions were seen, some of them being more or less localised in character, and yet others being generalised. The organs of the body infected have been various, and include the middle ear, the appendix, joints, subcutaneous tissues, pelvic organs, etc. Let me relate to you a few of those which are most interesting, so as to illustrate the kind of case that has come under this form of treatment.

Case 1.—The main interest in this case depends on the fact that the patient recovered from one of the severest forms of acute streptococcal infection, namely, puerperal septicæmia, the micro-organism being demonstrated in the blood-stream on more than one occasion. Moreover, metastatic abscesses developed during the course of the illness, rather larger doses of vaccine, prepared from the culture obtained from the blood, being given than is my custom. The dose used was forty million dead cocci. After the inoculation of the first dose the rigors which had been previously present ceased, and the temperature commenced to fall, although not in the striking manner noted in the erysipelas cases. The patient eventually got quite well.

Case 2.—A nurse, during the course of her duties, developed an attack of acute otitis media, which went on to suppuration. The pus was evacuated by an incision of the tympanic membrane, but this proving inefficient in the alleviation of the symptoms, the surgeon under whose charge she then was performed a radical cure on the middle ear and mastoid antrum. The patient's condition did not improve, however, although, according to the clinical signs and symptoms, there was no evidence of the involvement of deeper structures. An investigation of the blood was made, and the presence of streptococci of virulent characters demonstrated in the blood-stream. Up to this time the condition of the patient was causing some anxiety owing to the rise in the temperature, which was progressively getting higher. It was therefore thought to be advisable to try the effect of a stock vaccine, which was given in a dose of five millions. At the same time horse-serum was given by the mouth. Synchronously with this the temperature fell to normal

within the next twenty-four hours and remained there. More remarkable still was the change which was noted in the general condition of the patient, which very rapidly improved. The ear speedily healed.

Case 3.—This was a still more remarkable case, because virulent streptococci were demonstrated in the blood-stream of a small boy, æt. 4 years, and he subsequently recovered. Some month before this investigation was carried out the tonsils and adenoids had been removed with the usual precautions, followed by the customary after-treatment. For a fortnight he remained fit and well, but then he began to show signs of illness. His mother noted that he was a little drowsy, and at the time of examination he was found to have a raised temperature and rapid pulse.

It was at first thought that he might have got some form of meningitis, but efforts to prove this failed, and it was not until the examination above mentioned was carried out that the true state of affairs came out. The presumption was that the infection was taking place from the raw area in the naso-pharynx, although there was no purulent discharge and cultures failed to show that the coccus was present. Small doses of the autogenous vaccine were given and the boy recovered, the alteration in the course of the temperature chart and in his general condition being apparently synchronous with the initial inoculation of the dead cocci.

Case 4.—The patient was a surgeon who had infected his finger with a pneumococcus, presumably during an operation. This within a few hours was followed by a severe lymphangitis, spreading up the arm and requiring numerous incisions. This went on for five days without causing the temperature to fall. A blood-culture gave a negative result. Fortunately, however, a culture had been made from the initial focus of infection, and from this a vaccine was prepared and injected into the subcutaneous tissues. Synchronously with the introduction of the vaccine the temperature, which had been continuously raised for five days, fell to normal within twenty-four hours, and remained in that situation.

These few cases will serve to illustrate the type of infection that has been dealt with.

Excluding the cases of erysipelas already mentioned, twenty-one cases of acute streptococcus infection have been treated by vaccine therapy. In five cases the *Streptococcus pyogenes* was demonstrated in the blood-stream during life; of these, three recovered and two died. One case had streptococcus pyæmia following scarlatina and recovered. Two cases died with bacteriæmic symptoms, but no proof that such was the case was available, as micro-organisms could not be demonstrated during life, and post-mortem examinations were not made. Of the remaining cases, all had severe toxæmic symptoms but no evidence of bacteriæmia. Two died, and the remainder recovered from their various illnesses.

In discussing the treatment of acute streptococcus infection by these agents let me state certain factors which I consider to be tne cardinal rules of bacterio-therapy.

The first is that an accurate clinical and bacteriological investigation is an essential, not only as a means of finding out what is the causal agent of the disease, but also to obtain some estimate of the degree of the infection. The value of the blood-culture method has been alluded to in the cases cited.

The next most important rule is that proper efforts must be made to remove diseased tissues wherever possible, *e.g.* to open and drain an abscess, for without such there can be no advantage in introducing a vaccine with the object of effecting a cure. Vaccines should only be used as adjuvants and not as usurpers of surgical methods. If it is impossible to localise the focus of infection then vaccines become the only agents for treatment which we have at our disposal.

Having reached this stage, the next step is to institute vaccine therapy as soon as the bacteriological investigation has led to the isolation of the micro-organism causing the infection. To delay their use until the blood is already flooded with living bacteria is only to court disaster. In the early stages combination with specific sera may be found to be of some advantage.

With regard to dosage nothing very definite can be stated, but it is a good rule to say that the more acute and the more generalised the intection the smaller should be the dose of dead cocci introduced. The type of vaccine is also of some importance. Autogenous vaccines are the ideal agents for use in these cases, especially when one realises that the various brands of streptococci which may be met with are very numerous, but if, owing to the absence of a local focus of infection, or to the fact that complicated pathological investigations will be required to isolate the infecting agent, then stock vaccines prepared from virulent cultures of the streptococcus commonly known to be the cause of a given disease can be used, and with advantage.

The changes noted are similar to those described shortly in the cases of erysipelas, and for the most part some good can be said to have arisen out of the use of vaccines in these cases. One cannot go further from such a limited list, and one must remember that cases of streptococcus infection do get well in most remarkable ways, sometimes without the intervention of specific agents, but when such a recovery can be shown to coincide with the introduction of such agents, then the hopes for the future welfare of patients suffering from acute streptococcus infections must be brighter. The experi-

ence obtained from the use of this method of treatment in twenty-eight cases leads me to think that this hope may be realised.

Dr. HORDER said: Setting aside enthusiasm and preconceived notions as to what a remedy ought to do for a patient, no argument is so difficult in medicine as the therapeutic argument. It is at present quite impossible to adopt the statistical method in estimating the value of vaccines in the treatment of diseases. It should be remembered that in the use of a remedy for which a specific effect is claimed there is usually a tendency to expect a positive result. Various observers can do little else than state their general impressions; but if these impressions are carefully made, and are freed as far as possible from bias, they are very valuable.

When Wright first popularised bacterial vaccines in the treatment of infective processes I began to use these agents very extensively. My general impression as to their value was quite soon established past any doubt, and continued experience has but confirmed this impression. I am as certain as it is possible to be in such a matter that by the use of vaccines I have hastened a large number of recoveries from some infective processes, that I have produced a rapid change in the course of others, whereby the balance of resistance was disturbed in favour of the patient, and that I have now and again saved life when the patient seemed certainly doomed. I have never believed that vaccines were a panacea for all infective conditions, nor have I regarded them as more than a part, though at times I have had reason to think them by far the most important part of the treatment. I do not think I have ever made a patient worse by the employment of vaccines, although, as I shall presently admit, I have long ago ceased to control my dosage by the official method, without which it was formerly said that the treatment was dangerous.

In the short time at my disposal this evening I propose summarising my experience somewhat, and therefore I shall speak in generalities rather than weary you by the iteration of actual cases. My general statements will, all of them, be based upon actual cases. I will divide my remarks into certain sections, and will deal with (1) *prophylactic inoculation*; (2) *the choice of cases for curative purposes*; (3) *the question of dosage*; *and the* (4) *outlook for the future.*

(1) *Prophylactic inoculation.*—Into the question of preventive inoculation against *typhoid fever* I will not enter. This matter is almost the only one connected with vaccine administration that has attained to the dignity of the statistical method. There has of late been considerable activity in the preventive treatment of *influenza*, *common cold*, and *acute sore throat.* When faced with the question of protective inoculation against influenza, the first consideration that arises is whether or not the causal microbe of uncomplicated influenza has as yet been isolated. It is generally assumed that influenza is due to Pfeiffer's bacillus, but it may well be that this is but the most constant secondary infective agent in the disease. The *pneumococcus* and *B. catarrhalis* are commonly found in association with Pfeiffer's bacillus. To immunise against influenza by inoculation with Pfeiffer's bacillus carries a large presumption, and to promise a successful issue to this venture requires much boldness. But such an experiment, if carried out on a large scale, would be very valuable. Some immunisators inoculate against influenza by a mixture of vaccines derived from all three of the microbes just mentioned—a frank confession of doubt as to the specificity of the first-named. But the procedure seems rational, seeing that prolongation of the attack and the ill-effects of the disease are in great measure due to the secondary infections that are so common in it. My own plan is to await an attack in a susceptible person, and to investigate the flora of the upper respiratory tract during the first twenty-four to forty-eight hours. A vaccine is then prepared for service during this attack (if prolonged) and against future attacks. This method has given very promising results.

I adopt the same line in preventive inoculation against *colds.* I obtain an expert quantitative estimation of the flora of the nasal mucus early in the course of the illness and proceed accordingly. It is quite common to find the dominant micro-organism quite other than is anticipated. I therefore regard the use of a mixed stock vaccine in the present stage of our knowledge as bad practice. I have had experience of preventive inoculation in several cases of *sore throat*, in most instances streptococcal in nature, in persons with a marked tendency to periodic recurrences, and can report some very satisfactory results. In these cases the use of an autogenous vaccine is essential, as the strains of streptococci vary so much.

(2) *Choice of cases for curative purposes.*—If I were to pick my case—without for the moment considering the nature of the infection—I should say, Let it not be too acute nor too chronic. It is the subacute cases that appear to me to give the triumphs of vaccine therapy, cases that have attained to the point of unstable equilibrium, and where the stimulus of the antigen is enough to determine a change in resistance in the right direction. This may explain a fact which seems very striking, that in the cases that respond well to vaccine treatment it is so often a single dose, or a couple of doses of the vaccine, that effects the change. The mechanism of resistance in these cases appears too sluggish for any sharp response, and the attack of the micro-organism is not sufficiently virulent to lead to rapid effects; a balance

is present which can somehow be disturbed in the right direction by a dose of vaccine. In the cases in which, by long habit, the infecting agent becomes, as it were, saprophytic, little or no good effect follows vaccine treatment. This is seen very well in cases of colon bacilluria (I am not speaking of cases of cystitis or pyelo-cystitis). I have given up hoping to cure such cases, and make a candid statement of my experience to patients with this dose of vaccine as a prophylactic measure, and condition. I advise them to have an occasional dose with the idea of protecting them from spread of their infection, but I do not think they ever get cured by this method of therapeutics.

I have come to regard the actual infecting agent as a less important factor in determining whether or not there will be a successful issue to vaccine therapy than the nature of the disease and the resistance of the patient. Thus, after a great deal of consideration I cannot say that cases of infection by any particular microbe will do well, or of another will do badly, in response to vaccine treatment. And I am fully convinced that the present tendency to speak of diseases in terms of the bacterium associated with them is a fundamentally bad one, because it assumes that the microbe is the only factor of moment in the process. We hear that so-and-so is suffering from "*B. coli*," or from "streptococcus poisoning," as though these were diseases rather than the names of bacteria. We need to know to-day quite as much as we ever did the seat of the infection, the nature and extent of the lesion, the degree of resistance made by the patient, and the various somatic changes resulting from the interaction between the infecting agent and the tissues. And all this means careful clinical and pathological investigation.

It appears to me to be too readily assumed that the injection of specific bacterial antigen covers the whole field of a patient's immunity. I think it is much more likely that it covers but a part, and that it still leaves other factors untouched. If these other factors happen to be more important in a particular case the use of vaccine will naturally be disappointing. I am not one of those who thinks that anti-sera have no longer any function now that vaccine therapy is so fashionable. I regard immune sera as of great service when administered promptly in cases of acute streptococcal and pneumococcal infections, and I still employ them in these cases, combining with them often the use of small doses of vaccine. A better method may, of course, be the use of sensitised vaccine, as suggested recently by Besredka.

(3) *The question of dosage.*—I will admit at once that I take experience as my chief guide in this matter. Fallacious as this guide is known to be, I consider it more useful than the guide which is said to control the inoculations of many who study the question—that is, the opsonic index. Whether it is that this opinion has come to be shared by a growing number of workers, whether the hypothesis upon which the opsonic index rests is now regarded as more improbable than formerly, or whether, as is more likely, a determination of the opsonic index has been found unnecessary for the purposes of actual practice, need not be dwelt upon. It is quite certain that a very large amount of highly successful inoculation goes on to-day which is quite uncontrolled by opsonic determination. It is equally certain that these empirical inoculations are very frequently undertaken by workers who formerly taught that such proceedings were highly dangerous and were to be strongly deprecated. Wholesale chemists are provided with stocks of vaccines to be retailed for more or less indiscriminate use by those who formerly insisted upon the necessity for opsonic determinations. We may take it, then, that the facts of vaccine therapy have far outgrown the hypothesis that at its inception accompanied it and fathered it. This is a step in the right direction. I have been able to show, in a large series of animal experiments, that the effective range of dosage of bacterial vaccines is fairly wide, and that within this range no ill-effects are likely to ensue. Of greater importance than these observations on animals is the fact that in man the careful employment of vaccines, uncontrolled by opsonic determinations, has not led to any of those dire effects that were predicted for it. On the other hand, I have several times seen such temporary exacerbations of the infective process as do occasionally appear under vaccine treatment, manifest themselves during the most expert control by the opsonic index—one of several facts which long ago convinced me that the opsonic index, whatever its value may be in matters of diagnosis, is wholly unreliable as a measure of the patient's degree of immunity.

One of the great difficulties attending the question of dosage of bacterial vaccines is the *variation in virulence* of different strains of the same microorganism, and of the same strain after artificial culture. Particularly does this difficulty apply to various strains of the *streptococcus* group, to the *pneumococcus*, and to the *gonococcus*. It is very doubtful if this difficulty is sufficiently recognised by those authorities who claim that the opsonic index gives a correct guide to the amount of vaccine (bacterial endotoxin) which should be employed. Tables of suggested doses, say of killed gonococci, which are recommended by an observer for use in particular cases, lose much of their value on account of this important fallacy. Experience shows that whereas in the case of one strain a dose of five million dead gonococci produces a marked local reaction at the site of inoculation, and is followed by an exacerbation of a urethral discharge, in the case of another strain neither of these effects follows the use of a dose of

a hundred million dead cocci. Another difficulty, though apparently a less disturbing factor than that just named in the adjustment of dosage, is the individual differences seen in the response made by various patients to the same dose of the same strain of a micro-organism. This difference is often seen in the simple case of prophylactic inoculation against typhoid fever. Three persons may be inoculated with the same "stock" vaccine at the same time; one will show very little local or general reaction, another will present a moderate degree of both, a third will suffer from a red, swollen, and painful arm, with considerable malaise and pyrexia. If in the three persons the curve of the opsonic index shows the same characters, the mechanical school of pathologists concludes that the same degree of the immunising process has occured in each. But in the face of the differing degree of response as judged by clinical observations, this conclusion cannot be accepted without some criticism. A similar position exists, in a more complicated fashion, in regard to many cases of curative inoculation.

In the initial stage of this form of therapeutics, beyond which we have not yet passed, it is not surprising that great differences of opinion and of practice exist in regard to dosage. It does not, however, follow that even when our experience of the treatment is much greater than at present the question of dosage will settle down into one fixed rule which all will observe. Despite the many observations that have been made with regard to the use of anti-diphtheritic serum, opinions still vary as to the optimum size and frequency of dose; but the results of this therapeutic measure are, in the main, universally good. And in this instance, be it observed, there is no existing hypothesis which claims to control the question of dosage.

(4) *The outlook for the future.*—I have already said that I regard the altered position of the vaccine question in relation to the opsonic index as an advantage. I have for a long time held that its complete severance would be a greater benefit still, as this dissociation of the fact from the theory would dispose of the view, still largely held, that vaccine treatment is only for the rich and for the interesting case in hospital, because its employment necessitates elaborate laboratory investigations, and repeated supervision by a trained opsonist. Once the windows are thrown open, and a larger view of the scope of the method is taken, we shall begin to know the real limits of the treatment and how best to apply it. This will lead more quickly to what I regard as an inevitable position in the future: *vaccines satisfy one, and one only, of the requirements of immune therapy.* They do not form a complete system of treatment of infective conditions, valuable though they are in individual cases. They mark one mile-stone passed on the slow but certain march of progress in our struggle against the invading microbe.

Dr. BUTLER HARRIS, referring to Dr. Hale White's cases of infection with the *Bacillus coli*, said: My experience with *coli* infections of the urinary tract has on the whole been satisfactory. Certainly the method of treatment by bacterial inoculation is better than by washing out, and the giving of urotropin. Dr. Hale White mentioned the case of a woman who was a failure, where the kidney had been primarily damaged by a calculus. I can place alongside this case, that of a man who was operated on three years ago for tubercle of the right kidney, and who, some months later, was evidently going downhill from a chronic toxæmia. There were *coli* bacilli in the urine, and in the virus. His index was persistently low. Inoculations of an autogenous vaccine given at first bi-weekly and afterwards once a week for several months eliminated the *coli* bacillus from his discharges. His improvement was remarkable. His colour returned, he put on weight, and was able to return to business. It has, however, proved necessary owing to a return of the *coli* bacillus from time to time to give him inoculations. At any rate in this case, as in many others, it would seem that the patient requires occasional inoculations in order to maintain him in a decent degree of health. Dr. Hale White recorded also an interesting case in which the *coli* bacillus appeared to have infected the whole alimentary canal, a washing of the stomach containing *coli* alone. A parallel case in my experience is that of a small boy, æt. 6, who since babyhood has been liable to violent gastric intestinal attacks. He was dieted and drugged to no purpose. I found he was occasionally passing quantities of mucus by the bowel. This mucus was crammed with *coli* bacillus; and the exhibition of an autogenous vaccine given weekly in doses of five to seven millions completely cured him. The treatment was left off. Recently, however, there has been a return of the colitis with a much larger and more irregular coliform organism in the mucus. There is, however, no vomiting. A new vaccine is now being given.

It is interesting to note in this case that his mother is sallow and fragile, and her appendix was removed several years ago for recurrent appendicitis. One sister died after operation for appendix abscess, and another sister has recently been operated on, also for appendix abscess.

It is suggestive that heredity may manifest itself in the lowered resistance to a particular type of organism, and may be an important factor in what we are pleased to call vaguely "constitution." I noted with interest Dr. Hales White's practice of increasing the dose in the treatment by bacterial vaccines. My own experience has not been quite

so happy, particularly with *coli* vaccines. I have found on more than one occasion that even twenty millions have produced a rigor, a sharp rise of temperature to 104° F. with intense headache and joint pains. It, however, appears that so profound a negative phase is generally followed by a more marked and more lasting improvement than with the smaller doses. It is, however, in practice unwise to speculate so heavily.

As regards the *coli* tribe, I understand that many varieties are eliminated in the fæces. It is therefore a matter of some difficulty to ascertain, or even to be sure, of growing the particular variety or varieties which are pathogenic. I call to mind one particular case of colitis in which an eminent bacteriologist isolated thirteen varieties, did the opsonic indices to all of them, and then was at a loss to know which was the offending strain, as the indices varied. I am under the impression that it is exceedingly difficult to find a normal to any *coli* bacillus, and therefore it becomes necessary to pool a large number of bloods for the purpose of obtaining a reliable control. I am inclined to think that the agglutination test is more reliable.

To eliminate the difficulties in obtaining the pathogenic variety of *coli* bacillus I have found it more expeditious to obtain plate cultures from the washed mucus, when this can be obtained.

I quite agree that in acute infections, if there is going to be a response, it will be at once. In subacute or semi-chronic infections one must look at the problem from a rather different standpoint.

It always appears to me thus: The acutely infected person is stricken down by some unforeseen blow; it is an accident of nature as it were. Hence if the invasion is not too overwhelming it is reasonable to argue that he will respond to the stimulus of inoculation. The individual labouring under a chronic malady is like a man who is carrying a heavy load, the weight of it and the ease of carrying it varying with his resisting power, a load which in few instances can be thrown away on the journey through life. These cases, I feel sure, as our experience lengthens, will only maintain a reasonable degree of health by leading a strictly regulated life and having recourse from time to time to series of inoculations.

To illustrate with the same organism—the pneumococcus:

A— has an attack of acute pneumonia, from which he may recover by auto-inoculation and a normal crisis, or early inoculation and artificial crisis, or lysis.

B— has had a pneumococcus present for many years in his naso-pharynx. He suffers from severe colds in the head, bronchitis, and at times is so poisoned that his general health suffers, and he is unable to follow his occupation. He has had every form of nasal spray under the sun, and only returns to a normal, even line of health when inoculated systematically with his own pneumococcic vaccine. He remains well for six months. Overwork, and an infection of Friedlander or catarrhalis, lower his resistance, and his old symptoms return. He actually writes lately, "I believe my old enemy has returned." A smear is seen to be crowded with pneumococci, which for months had disappeared. A further course of inoculations are demanded to the first one. He responds at once.

There is little doubt that cases like these must expect to fare in this way, and, like those infected with tubercle, should be always armed against a future invasion.

When we have failures in this work it is often because we have not got the right organism, or because the organism has its chief *depôt*, so to speak, in some damaged recess in the body, which is un-get-at-able, or has not been properly cleared out by the surgeon. I note that Dr. Girling Ball, in the rules which he formulated at the end of his paper, laid stress on the necessity for the removal of diseased tissues and the opening of abscesses. And there is no doubt, as Wright has pointed out, that Bier's suction method and his treatment by hyperæmia not only gets rid of diseased material, but, what is more important, bring fluid rich in protective material to the areas of low bacteriotropic resistance.

The following case is a very clear illustration of this principle.

A lady had had recurrent attacks of facial erysipelas for over two years. These were becoming more frequent, and were induced by over-fatigue, or exposure to cold winds. The inflammation always started in one spot—on the junction of the cheek and nose about half an inch below the inner canthus of the right eye. She exhibited a fairly low index to the *Streptococcus pyogenes* at the beginning of an attack, which rose to about 1·2 towards the end and then gradually fell to normal. The general constitutional disturbance was little, the local inflammation decided. Inoculation with five million stock streptococcus vaccine caused a sharp rise in her index, and this was maintained as far as possible about 1·5 by weekly inoculations. The local benefit was not, however, so marked as one would have expected, nor were attacks wholly warded off. There was no doubt that the cumulative effect of many attacks all starting from the one tiny focus had damaged the circulation in that particular area; and therefore it was reasonable to argue that here was an area of more or less constant low bacteriotropic pressure, although the protective content of the rest of the body was high. A daily mild cupping with a hot liqueur glass was accordingly instituted. The result was so satisfactory that not only did the attacks cease during last summer, but this winter the patient was able to return to her ordinary out-door life,

and has hunted all the winter with impunity. As a safeguard she has still occasional inoculations.

My belief is that now the few streptococci which remained behind after each attack in some secure recess unwashed by high potential serum were dislodged and destroyed by the daily flooding produced by this simple method of obtaining a local hyperæmia.

I have previously published a case where there was a disfiguring patch of acneiform eczema in the face, which was similarly cured. And as it is with these small infections, so must it be with the larger and more serious ones which are deep-seated, such as *coli*, or tuberculous or gonococcal infections of the kidney or prostate.

I think this is the explanation why the mucous membrane, and tissue infections, without gross destruction of tissue, yield so much more rapidly to inoculation than those where the vaccine needle has been substituted for a free use of the knife.

Early tuberculous joints, such as hips, do very well with rest and inoculation; but later types require just as much active surgical treatment as of old; but the healing is undoubtedly accelerated by appropriate inoculation. A lad under the care of Mr. Tubby was operated on for pelvic abscess. There were several sinuses leading to the pubic bone and the ilium. The pus contained a large intestinal diplococcus, which was isolated and a vaccine made. After free drainage had again been established the wounds rapidly responded to the inoculations, though for some time they had resisted all attempts at healing.

As regards bacterial inoculation for pneumococcic infection, I may say briefly that a pneumococcic vaccine made from a virulent strain will more often than not produce in pneumonia a reduction of temperature by steps, it being necessary to inoculate with twenty to fifty millions every other day. The treatment fails when the toxæmia is very profound and the whole circulatory apparatus is paralysed by an overwhelming blow. It is best to use an autogenous vaccine, and Willcox, of St. Mary's Hospital, finds that the pneumococcus derived by drawing blood with a hypodermic syringe direct from the consolidated lung yields a better vaccine usually than that obtained from the sputum. My own experience is that the pneumococcus from a virulent case grows very rapidly on blood-agar, so that it is possible, in spite of what has been asserted to the contrary, to make sufficient vaccine within twenty-four hours. To save time I would refer the Society to a paper on pneumococcus inoculations published in the 'British Medical Journal' for June 25th, 1909, from which I will quote the conclusions. Since this publication Willcox and Parry Morgan read a paper at the British Medical Association meeting at Belfast citing some twenty cases of pneumonia, most of which were successfully treated by a bacterial vaccine. Dr. Willcox informed me a few days ago that a stock vaccine made from about half a dozen stains was now put up in the Inoculation Department of St. Mary's Hospital, and was being retailed by Parke Davis & Co. As it is all-important that the vaccine should be given as early as possible in pneumonia, there should be now no difficulty in obtaining it.

With regard to the sequelæ of pneumonia, there is no doubt whatever that inoculation is generally effectual. Pneumococcal injections of joints, unresolved patches in the lung, phlebitis and thrombosis usually do well when all other methods of treatment have been tried and have failed.

The *rôle* of the pneumococcus in producing severe recurrent catarrh with fever is, I believe, not usually recognised. One case I have alluded to earlier. Another interesting one was that of an Indian civil servant invalided home with supposed recurrent attacks of malarial fever. I say "supposed" advisedly, because I ascertained that his blood had never been examined for the malarial organism.

I examined his blood during three attacks last summer and failed to find it. I did, however, notice that each attack was accompanied by severe nasal and bronchial catarrh, and that during the attack his secretions were swarming with pneumococci. Inoculation with a stock vaccine during an attack at once cut it short, and a systematic course of this treatment prevented the occurrence of more, until after six weeks' respite from inoculation a small one occurred. He was, however, able to return to India in vastly improved health, and inoculating himself at regular intervals has remained perfectly well for three months. He had been a victim of these attacks for the past four years.

I think those who have practised bacterial inoculations will agree that, as in all other forms of treatment, the earlier the procedure is adopted the more likely is the patient to recover, and to recover quickly. Unfortunately it falls to the lot of the consultant who is called in to carry out this treatment to find generally that he is appealed to as a last resource.

I believe this is the result of the early training of the general practitioner. I am aware that much more attention is now being paid to teaching bacteriology, but I am convinced, after my own experience of sixteen years in general practice, that a working knowledge of the commoner pathogenic organisms is absolutely essential to careful work. I don't know whether it is the custom nowadays, but I would insist upon every clerk and dresser taking the materials from his patient for bacteriological examination to his bench in the laboratory, and there, under skilled supervision, working out for himself the infecting organism.

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CONTENTS.

PAGE

*A Clinical Lecture on Sigmoiditis and Proctitis. By F. C. WALLIS, F.R.C.S. 241

*Migraine. By GUTHRIE RANKIN, M.D., F.R.C.P. 247

*Some Practical Points in the Management of Difficult Labour. By HENRY RUSSELL ANDREWS, M.D., B.S., M.R.C.P. 251

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A CLINICAL LECTURE

ON

SIGMOIDITIS AND PROCTITIS.

Delivered at St. Mark's Hospital for Fistula.

By F. C. WALLIS, F.R.C.S.,

Surgeon to the Hospital and to Charing Cross Hospital.

GENTLEMEN,—The subject of my lecture to-day is sigmoiditis and proctitis. I want you to eliminate gumma and syphilis from your category of possibilities in sigmoiditis and proctitis. These are diseases of the rectum which it is necessary to recognise at once and to give the patient the necessary treatment. Owing to a mistake which has been made for many years in text-books, students are told that syphilis causes all sorts of diseases in connection with the bowel, with the result that various conditions are classified as syphilitic and treated as such. In that way valuable time is wasted. These diseases, as a matter of fact, are not syphilitic. I have not seen one case of syphilitic disease of the mucous membrane of the bowel or rectum since I have been attached to this hospital, and that has been a considerable number of years, in which time I have seen many thousands of cases. Here is a special hospital for diseases of the rectum, and people come here from all parts of the world. (Only this afternoon I was operating upon a man with carcinoma recti who was sent to me specially from Kimberley.) We see rectal and anal conditions in people from all climates. We also get a lot of Russian and Polish Jews. In the case of the man on whom I operated this afternoon for carcinoma it was supposed that he had syphilis. He had been treated with mercury and iodide of potassium for months, and of course the growth was allowed to go on during that time, and valuable time was wasted.

We must have some sort of classification for sigmoiditis and proctitis, and in the first instance they

are divided into acute and chronic. Then we will start with the statement that they are all microbic infections. Take the chronic: there is the dysenteric, which may be acute also; there is infective sigmoiditis and proctitis; and one may as well include gonorrhœal, though I have never seen a case of it. It is uncommonly rare, but it does occur. Unless you have absolute evidence, it is, of course, waste of time to treat a thing for what it may possibly be. I have put the dysenteric form first, because it is often a misnamed disease. A large number of men are sent home from India and subtropical regions with what is called dysenteric ulceration. I shall not refer to the infective dysenteric ulceration which occurs in asylums, because you know all about that, and, as a matter of fact, we are not dealing with that class of person. With regard to the so-called dysenteric ulceration which is sent home, I am bound to say that a large number of these cases may have started in some form of dysentery. Of course, the diagnosis of dysentery is getting much clearer now amongst most people, especially among the officers of the Army Medical Department, but, even so, there are still a large number of them who do not appreciate the difference between dysenteric ulceration and colitis, or sigmoiditis, or proctitis, which may arise from chilled water or bad water, and in which the amœba of dysentery does not exist. They are classed as dysenteric because there is an ulcerative proctitis associated with diarrhœa occurring in a tropical country. They come home, and are treated with ipecacuanha, and they make very slow progress. The question of a person's recovery depends on his ability to get up to a certain point of resistance, and then he overcomes this ulceration, which no doubt is of the infective variety. As to the diagnosis of sigmoiditis, the symptoms are these, and I am talking of chronic cases, because I want to leave the acute to the end of the lecture. The symptoms are pain, a rise of temperature, sometimes a rigor, blood-stained discharge associated with a certain amount of diarrhœa, and with very much mucus. There is a straining down, and a feeling of weight in the rectum. The whole of the pelvis seems to be involved, and the patient himself feels extremely ill. Those are the symptoms which he comes complaining of. The ordinary digital examination will not reveal much, except that the rectum is extremely hot. It also feels rather more velvety than ordinary healthy mucous membrane. You may have some difficulty in getting the patient to allow you to examine him with any thoroughness, because it is painful, so you may not learn very much from a digital examination. You can learn something by examining the man by means of the speculum. If it is proctitis, you see the extreme congestion of the mucous membrane. One cannot be surprised that the patient does not want the examination to be made. The proper diagnostic instrument is, of course, the sigmoidoscope. If you put cocaine into the bowel, and let it remain there for a few minutes, you can pass in the sigmoidoscope without much discomfort to the patient. If he does not allow it, then he must have a general anæsthetic. You would not, of course, have to press on the sphincters too much in examining such a bowel. A small electric lamp is passed down inside the tube and locked there, the current is attached to the terminals, and is turned on, and it enables you to see inside the bowel. If the man has not been properly prepared you will find there is a great rush of blood or blood-stained mucus, or fæcal matter, and this may have to be repeatedly swabbed out. Occasionally one may find that when the ordinary forceps are used a piece of cotton-wool is left inside the bowel. I use a stout wire, which is fenestrated at the end, and I wrap some cotton wool around the end, and that is put in, and after swabbing it can be safely withdrawn. If you leave a pad of cotton-wool behind it spoils your examination, but there need be no alarm about it. Here are representations of the various forms of sigmoiditis and proctitis as seen through the sigmoidoscope. Catarrhal proctitis represents congestion of the whole mucous membrane; it is congested and dirty with blood, and it is pushed forward in almost a prolapsed condition, and patches of inflammatory exudation are seen in the submucous coat. Atrophic catarrhal proctitis is one of Tucker's expressions. I do not know what it means, but I should say it is a kind of chronic proctitis. Instead of there being an acutely inflamed condition it has become a much more chronic condition. The mucous membrane is more or less purple in patches. In catarrhal proctitis there is also a condition of membranous colitis. This goes down into the rectum, which has a muco-purulent discharge, and on examina-

tion the mucous membrane has a peculiar velvety feeling. Follicular proctitis is a curious condition which I will not worry you about. It seems to last for a long time, and does not cause very much discomfort, and it is difficult to cure. There are little follicular pits dug out, they are almost punched out, and they bleed occasionally. In ulcerative proctitis the mucous membrane is rather inflamed, and there are considerable abrasions and regular ulcerated areas. That is the common condition, which we have to deal with as the result of dysenteric ulceration.

There are other forms of sigmoiditis which we have to bear in mind. The following is a case in point: I was called to see a young fellow about twenty years of age with an acutely severe pain in his rectum. There were all the indications I have already mentioned, including straining, and when we examined his bowel with the sigmoidoscope it was found to be intensely congested and the subject of acute inflammation of the rectal mucous membrane, the inflammation extending into the lower part of the sigmoid. I thought it was one of those horrible cases of a young fellow having a growth which had become rather inflamed. The man was so bad that we took him into Charing Cross Hospital, and with the idea of giving him relief we opened the abdomen in the mid-line to see what sort of growth it was, and whether a colotomy would be the best thing for him. I found that his appendix had become acutely inflamed and was adherent to the anterior wall of the sigmoid close down to the rectum, and this inflamed, infective appendix had produced all these symptoms. I mention this as a curiosity. If there is an acute onset in an otherwise healthy man you must not rush to the conclusion that his appendix is wrong; there must be very few cases of that description. But you may constantly find in the bowel something quite different from what you expected, causing peculiar symptoms. And I think the routine treatment was the best, because we had no idea what the trouble was, except that it was very acute, giving the man intense pain, and it was getting worse, so we thought it was a very actively progressing growth.

I have mentioned micro-organisms as a cause of these conditions, and I suppose the particular micro-organism which causes more ulceration than anything else is not the amœba of dysentery or the *Bacillus dysentericus*, but it is the *Streptococcus longus*, the one which is found about the mouth. I have over and over again seen chronic ulcerative conditions, sigmoiditis, proctitis, and colitis with this micro-organism as the cause. Take a sterilised swab and get a specimen for bacteriological examination from the mucous membrane of the bowel, and you will often find the *Streptococcus longus*. It is nearly always mixed with the *Bacillus coli*, and possibly with others also. In ulcerative proctitis and sigmoiditis the teeth should be carefully looked to. In hospital patients particularly you will find a very large proportion of them have very bad teeth, and probably they have had them so for several years, and they have been continually swallowing these poisons. Here we clean the mouth and keep the gums clear with peroxide, and by keeping up this treatment for several days we make the mouth much more healthy. Another common cause of these disorders is constipation. Of course I cannot go into that, but I should like to say that if anyone can tell me of a cure for constipation I shall be very glad. We know there are many causes for constipation, and we may be sure that constipation is a great factor in producing the conditions which we are now discussing. It is perfectly clear that if the patient has a large amount of accumulated fæces, passes a portion, but only a portion, at a stool, and the rest remaining for some days or longer, there must be a definite degree of absorption of this material into the blood, and in consequence of that the resistance of the individual will be lowered. In the very acute and fulminating cases the patient may die in twelve to fourteen hours. There is another thing that I want to say about these chronic rectal ulcerations, a subject which interested me for some years, and it is that a large number of these chronic ulcerative conditions, which go on for a long time, are produced by a small lesion to start with. I have watched such conditions from the smallest lesion to the most advanced cases. As you know, the proctodeum joins with the blind end of the gut, and where the junction occurs there is a puckering of the mucous membrane, which produces what is known as the crypts and columns of Morgagni. A very good illustration of them is to take a coat-sleeve which is very big and notice the puckers which occur in it. Now, given constipation or strain, or anything of that sort, there may readily

be a lesion formed there. A certain number of symptoms are thus caused which may make the sufferer seek relief. There is a submucous infiltration, which, bulging underneath the submucous tissue, gradually produces ulcerative proctitis. Healthy mucous membrane, as you know, is most resistant, and it takes a great deal to upset it. But if there is a lesion at the part where it is most likely to occur you may trace the onward course of the disease, and it is a horrible scourge when it does happen. There is an infective invasion of the submucous tissue. The loose areolar tissue around here is less resistant, and in this micro-organisms can multiply. Gradually the blood-supply to the part is cut off, and the mucous membrane is destroyed. Occasionally the mucous membrane looks like wash-leather, and again, at times you will find it injected, and it is rough and granular to the touch. I have followed these processes for years now. I saw two cases two and a half years ago which were most interesting in this respect, because in one individual I saw three regular submucous test-tube tracts, and in my small book you will see another picture of the same sort. You could put a probe into each one of these sinuses and squeeze pus down and take a culture from each pocket. These various pockets coalesce, and so gradually there is an increasing area of ulceration. The mucous membrane is raised and thickened and extends half an inch or more beyond the ulcerated area. This condition may go on, not for six months or a year only, but for a number of years. The first case I ever had and the one which led me to investigate into this condition was a child, æt. 6 years, who came from the West Indies. She was going round with a gipsy troupe, and some people who had no children of their own adopted her. They came over here, and this child was brought into my hospital with ulcerative proctitis, about which I did not know much at that time. She was an absolutely healthy girl in every other way, and her history of ulcerative proctitis dated back to six years of age. She had a discharge from the bowel, and her age when I saw her was 13½ years. So this condition had been going on for 7½ years. The ulceration involved 3½ in. of bowel, and extended round the whole circumference of the gut. Examination gave her considerable pain, and she had this constant discharge and also occasional attacks of acute painful swellings of the joints, which were due to infection. Whilst she was in the hospital her "adopted" parents abandoned her, and she was in the plight of being a healthy girl with a discharging bowel, unable because of this to earn anything. She had constantly to be changing her clothes because of this discharge, and could not go into any service. Things being so desperate with her I determined to excise the whole thing. It was the first time it had ever been done for this disease, and she made a very good recovery, though she nearly died as the immediate result of it. I have seen her at various times since, and if you examined her bowel to-day you would not know that she had had any operation done at all. It was a most instructive illustration of this appalling condition. If the patient is unhealthy, or not resistant to invasion by micro-organisms, the condition spreads much more rapidly. It is more common in girls than men. Sometimes you find the whole mucous membrane has gone and you can do nothing with them except perform colotomy, and it is a poor look-out for some of them even with that. That is the condition of chronic ulcerative proctitis, which can be anything from a small ulceration up to a very serious and fatal affair. Infective ulcerative proctitis may occur after an operation, and occasionally occurs after ligature of piles. It also occasionally follows operations for fistula. It is infective, and is due then to insufficient antiseptic precautions. In St. Mark's Hospital there used to be a condition known as "The Ulceration." It used to spread through the wards and compel us to close the hospital for a time. It was in the old days, when one patient was examined and the examiner passed on to the next, when the nurse dressed one case and then passed on to the next, without any discrimination as to what was the matter with A. and what was the matter with B. We do not see these cases in these days, but occasionally you see it commencing after an operation, such as ligature of piles. Such results have been brought to me from time to time and show chronic ulceration of the rectum. People with this condition have a salmon-pink discoloration of the mucous membrane. The treatment of these cases is fairly successful now. We have mentioned about the mouth and the micro-organisms which infest it: I would say again, Do not forget to examine the mouths of these people, and give them intestinal antiseptics. I suggest for this

purpose glycothymoline, which is admirable, or Beta-naphthol. Another good thing is to give some form of lactic acid bacillus: I have great faith in this for such a condition if you can get the people to take it. Of course the bowels must be emptied to start with by means of enemata and by means of proper purgatives, of which castor oil is the best. Lavage to the colon is a very good thing to carry out, and it should be done. It is a matter which every doctor should know how to do. I shall not speak of it now, because you can find it dealt with in any book. It should be done with a large tube, which can be passed almost to the cæcum, and the amount of water given should be injected quite slowly; three or four pints should be given, gently kneading the colon afterwards from the cæcum downwards. With regard to the local applications, I do not know that I think much of them. For the moment I am thinking of cases of rectal ulceration. They were taken out of hospital and put into homes, and sent for sea voyages and all sorts of things, but they steadily became worse and generally died after years of suffering.

I will revert to the condition of commencing infective ulceration occurring after ligature of hæmorrhoids. If you put these people immediately into a hot bath and let them soak in it, and wash and swab out the rectum with diluted oxide of hydrogen, you will find that the symptoms will be ameliorated, and there will be a yellow purulent discharge instead of a blood-stained. It is an indication that the acute symptoms are over, and you get what is called healthy pus, though I do not know what that means. But if you get rid of the salmon-pink discharge, getting instead the yellow pus, you will know that your troubles are over with regard to the progressive part of it, and you can get the patient well afterwards. In the treatment of these conditions, what Dr. Ironside Bruce and I have investigated, and I think with success, is ionisation, that is to say, ionic medication. We have an apparatus upstairs for carrying out cataphoresis. The details of that I shall not be able to give you now, but there was a paper on it which we published in the 'Transactions of the Royal Society of Medicine' last year. Certainly the effect of it is remarkable. There have been two or three cases recently in which one application of the method has cured proctitis, a condition which might otherwise have gone on for a number of months. One such case occurred to me, that of a woman who was sent into the hospital with proctitis, a very bad sinus, and a discharge from the rectum; there was also some pain and temperature. Otherwise she was healthy. She was the wife of a coachman of one of my colleagues. We gave her one sitting of ten minutes' zinc cataphoresis, and she never looked back, the whole of the symptoms disappeared, and she got quite well. That was a case which was caught fresh and young in a patient otherwise healthy. In chronic cases this has done wonders. I have had cases coming up week after week and month after month. We have cured most of them by means of zinc ionic medication, which cured the condition of progressive ulceration. It killed the micro-organisms, and repair seemed easy afterwards, making the people as comfortable as possible.

It may be necessary in some of these cases to do excision of the rectum, and that I have done in various instances. Very often the ultimate result of this ulceration is fibrous stricture. I do not know whether any of you have had to dilate these strictures constantly with bougies, but it is a very thankless task, and a painful one too, and I do not think very much of it. The stricture itself is usually situated about 1½ in. to 2 in., or 3 in. as an extreme limit, from the anus. You can excise the strictured portion and bring down the healthy mucous membrane in the ordinary way, and the results are very good. In other instances, on account of the advanced ulceration, you may have to do colotomy, because there is stricture six or seven inches up. I have a woman now under my care who has a stricture two or three inches long, and it is up in the sigmoid. We opened her abdomen and inspected it, and we found it would be a serious matter to attempt to excise the affected bowel. Therefore we did a colotomy, and it is extraordinary how patients get used to that treatment. We find such patients after this operation are well and healthy years afterwards. When sigmoiditis is getting up to the descending colon, and is therefore more or less chronic, and possibly merging into colitis, it will be found often that appendicostomy is a very useful operation. I shall not say any more about that now, because I think it is a matter which Mr. Mummery will deal with later on when he talks about colitis. In ulcerative conditions of the rectum and sigmoid, I think

the best operation to do is a temporary colostomy. I have done that now in four or five instances, and the results have been excellent. Two men I have in mind were perhaps the most interesting. They were both soldiers, both had been sent home with the statement that they had dysentery. One man had a very old hæmorrhagic ulceration or ulcerative process. He had lost a large amount of blood, and was getting very anæmic. The other man had not a hæmorrhagic condition, but a very marked ulcerative condition. In both those cases temporary colotomy was done, and by that means one can keep the affected portion of bowel absolutely clean. No fæcal contents pass over it. You can irrigate it and give it time to rest, and leave to Nature the healing process. When that has happened, and there is healing, you are in a better position to prevent any contraction by means of passing a bougie or the finger, or doing anything which you think is desirable in the way of operation and producing the best result. You have a comparatively speaking clean material to work on. With regard to the intestine, the colostomy wound is sewn up, and that is a simple matter, and most effective. And let me suggest to you that this question of temporary colostomy is one which should be more considered than it is in the treatment of these cases. It sounds rather like a refuge of the destitute, and very appalling, but it is really nothing of the sort. It is a good treatment, because instead of giving these people untold pain and continual distress, which they have every time the bowels act, or the contents of the intestine come down on the inflamed region, you give them the best chance they can have of getting better, much quicker than otherwise, and there is much less liability of the spread of the disease. And these patients are very much better able to fight against whatever trouble they have. Altogether the state of things is infinitely better than it would be if you did not do a colostomy.

Now one word about the acute condition, because you should know about it. Acute conditions are not very common here, but if you have seen these acute suppurative infective proctitis cases, or acute infective sigmoiditis cases, you will agree with me that they are most appalling. I have never seen acute proctitis or sigmoiditis of a fulminating character in which death occurs in a few hours in a woman. I have only seen it in men. But this may be coincidence. The man comes in with the history that he has been feeling ill for months past, that he has been passing blood, and has had some diarrhœa and so on, and he looks ill. The pulse-rate is found to be rapid, his temperature is high, and you examine the bowel and find it is intensely painful, and that it feels very hot. On examining him *per rectum* you probably find a gush of foul-smelling filthy material, blood-stained and intensely stinking. Probably with that a slough will come away also. In general appearance you will find the man is hollow-eyed, that his cheeks are sunken, and his abdomen retracted. After taking food, even milk food, his bowels act quickly. On handling the abdomen it is found to be very tender, and the man has the appearance of a person who has been made extremely ill by poisoning. The next case of this kind I have I shall propose to do an appendicostomy at once, and wash through it as many pints of fluid as I can get through. I should do that with some antiseptic such as glycothymolin. At the same time I shall perhaps transfuse. I have not gone into those details much here, because this is a matter of urgent operative interference, whereas my purpose to-day was to speak of every-day chronic conditions which you may come across in connection with inflammations of the sigmoid and proctodeum. These conditions are extraordinarily interesting from the point of view of diagnosis, the earlier you diagnose them the better you get them affected by treatment. If they are not diagnosed early and you have to treat them in the later stages, you will find it is difficult to get them well. I wish particularly to recommend to you the ionic zinc medication, and also temporary colostomy in certain conditions of ulceration of the sigmoid and rectum.

July 25th, 1910.

Palpation of the Abdomen.—Galambos depresses the abdominal wall with one hand, pushing the furrow thus made toward the organ to be examined. This relaxes the wall between the organ and the furrow so that the region is soft and more readily palpated. The hand must be pushed down gently into the depths, and the entire thickness of the wall worked down toward the organ in question. It is possible in this way almost to turn over organs to facilitate palpation.—*Journ. A.M.A.* vol. lv, No. 2.

MIGRAINE.*

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THERE are very few of us who have been long in practice before being called upon to deal with that prevalent and often intractable complaint known popularly under the name of "paroxysmal sick headache." Though, because of its prevalence, it is a disorder which may be classified among the commonplace ailments of humanity, and, because of its characteristic phenomena, one of those which offer no diagnostic difficulties, yet none the less its efficient management is a practical problem which faces us on the very threshold of our professional experience and upon the successful solution of which may depend the acquisition of the first step in the reputation upon which our life-work is to depend. I therefore make no apology for choosing migraine as the subject of this afternoon's lecture, because although I am unable to tell you anything new about a disorder concerning which so much has been written and spoken, I may succeed, by reviving in your memories the prominent characteristics of the disease, in stimulating your interest in some of the interesting questions which arise from a short consideration of the "why and wherefore" that cluster round its ætiology and treatment.

Migraine is undoubtedly an hereditary ailment, and we meet with it, as a rule, in families with pronounced neuropathic tendencies, not infrequently associated with gouty proclivities. It is important to remember that, though it most commonly first declares itself about the age of puberty, it is by no means unknown in early childhood, and may, though more rarely, first declare itself at, or even after, middle life. Both sexes are liable to be affected, but women suffer more frequently than men, the upper and more highly educated classes being specially vulnerable. Of the immediate causes of a paroxysmal headache there are three which stand out with conspicuous prominence: uncorrected errors of refraction, disturbances of digestion, and mental worry. But these are extraneous and accidental incidents only which are to be regarded as mere exciting causes of a symptom-complex which seems to be due to some congenital defect in the inhibitory mechanism of the brain, often associated with some fault in the chemistry of nutrition. In its initial manifestations, the most characteristic phenomenon is one variety or other of visual abnormality: Flashes of light, blindness of one half of the visual field, a small circle of sparkling colours, a curious zig-zag spectrum of a fortification pattern, transient hemianopia, are among the most usual subjective prodromata of an attack. These may occur in combination with other sensory signs, such as tingling in the limbs, face, or tongue, temporary aphasia, or slight vertigo. Smell, taste, and hearing are seldom or never involved. In some cases temporary paralysis of one or more branches of the third nerve has been observed, usually on the side which is affected by the pain. Sooner or later, after these preliminary warnings, and generally as they are declining, there ensues headache of varying intensity, which generally lasts for the greater part of a day, and increases gradually until, when it has reached its maximum, it is partially relieved by active vomiting or retching, and thereafter gradually subsides under the influence of a sound sleep, to which the sickness is usually a welcome prelude. In most cases the headache is unilateral at first and occupies a circumscribed spot on the temple opposite to the eye which was mostly involved in the initial spectrum, but in the course of the attack the pain may shift from one side to the other, or the whole frontal area may become involved. It is of a peculiar boring character, and is so aggravated by noise, light, or movement that the patient, when the attack is severe, cannot bear to be interfered with but lies in bed in a semi-stupid condition, and begs only to be left alone and in the dark. Appetite is wholly wanting and the vomiting causes intense prostration. Occasionally diarrhœa coincides with the vomiting. During the attack the urine is scanty, of high specific gravity, and generally loaded with phosphates. The headache is sometimes accompanied by a subjective feeling of heat which is often limited to the head and neck, and is associated with coldness of the extremities and a sensation of shiveriness over the surface of the trunk. There is always a certain amount of legarthy, and sometimes a degree of mental confusion which prevents the proper expression of

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thought, and may go the length of temporary incoherence of speech. Vaso-motor symptoms are often present; the face, for instance, may be at the commencement of the attack abnormally pallid and cold, while later on it becomes no less strikingly crimson and hot. The occurrence of one attack predisposes to others which happen at varying intervals, and seem often to be determined by special circumstances, such as an unwise meal, the occurrence of menstruation, mental upset, or physical fatigue. The disease may last throughout life, but it often disappears spontaneously, and in cases where it dates from an early age it may vanish after the climacteric in women or after middle life in men. It is interesting to note that migraine, in some instances, co-exists with other neuroses such as epilepsy or asthma, and that as a rule whichever disorder first existed tends to become diminished or entirely replaced by the newcomer. It has been known to disappear permanently on the occurrence of an attack of acute gout.

No coarse anatomical lesion has been found to account for the disease, but it is fairly certain that the primary cause of the attacks exists in some disturbance of function in certain parts of the cerebral cortex causing a nerve-storm from sensory centres, similar to the periodic discharge of energy from motor centres which is probably accountable for epilepsy. Whether the true explanation is a functional neurosis or a toxæmia to which the cerebral symptoms are secondary is still a question about which there is a conflict of opinion. The toxæmic theory derives some support from the fact that sufferers from the disease frequently have a pronounced gouty history. The combination of visual and other sensory disturbances followed by headache, which, at least in the first instance, is hemicranial in distribution, and which terminates in nausea or active sickness, together with the periodic recurrence of the same sequence of symptoms, and a family history of migraine or some other variety of nerve disorder in previous generations, presents a picture which cannot be mistaken for any other form of headache. The diagnosis, therefore, is easy and unmistakable except in a first attack, when it may, reasonably enough, be mistaken for a simple headache dependent upon a passing upset of gastric or hepatic origin. It may be pointed out, however, that when migraine first declares itself at or after middle life, or when at that age the prodromal manifestations present themselves in a new and unaccustomed form, the diagnosis may seem uncertain. If, for instance, the premonition involves sensory or paretic phenomena in the arm or leg, or impairs speech, or produces vertigo, and if there is little or no subsequent headache and vomiting, a strong suspicion is aroused that the patient is threatened with, if not actually suffering from, a vascular cerebral lesion. Any initial doubts will, of course, be cleared up by the subsequent course of events, but the possibility of a mistaken diagnosis in such cases should not be lost sight of.

This painful disease is as a rule very rebellious to treatment, but it does not involve danger to life, and, in the majority of cases ultimately, though it may be after many years, gets well spontaneously. Treatment divides itself into two stages, that in the intervals between the attacks, and that in the currency of the attack itself.

During the interval the first thing to be attended to in every case is the condition of the eyes. When it is remembered how often refractive errors are associated with migraine, it will be at once realised how important it is, as a prophylactic measure, to have such errors, if they exist, corrected at the outset of treatment. The next point of importance is to devote a strict attention to the diet, which should be simple, nutritious, and of moderate amount. All forms of rich food must be avoided; sugar should be taken sparingly; red meat once a day only; tea in very limited quantity; and salads, with all varieties of uncooked vegetable, not at all. Alcohol is best done without, and tobacco ought to be indulged in in strict moderation. Diluents should be taken freely, and a large tumblerful of piping hot water first thing in the morning, and again before going to bed at night, is a useful means of keeping the tissues free from accumulation of waste products. The fact that migraine affects mostly those who live sedentary lives and passes by those who are active and constantly engaged out-of-doors gives us a hint as to the necessity for exercise of a suitable kind in the open air. All kinds of active out-of-doors games should be encouraged both in summer and winter. A tepid bath, followed by active towelling, should be a matter of daily routine, and, in those who are vigorous and active a weekly Turkish bath is often

of great service. The bowels always demand careful attention, and when constipation is a source of trouble it may be corrected by a morning dose of Carlsbad salts, or Apenta water. Once a week the patient should indulge in an aperient of an alterative type. A pill consisting of one grain of calomel with four grains of rhubarb will be found useful for this purpose.

It is of first importance that the digestion should be kept in active and efficient order. To this end it is always essential to investigate the state of the mouth and to make sure that every dental fault is remedied. The existence of carious teeth is responsible for various forms of auto-intoxication to a far greater extent than until recently was believed possible. Not only should the teeth be overhauled and attended to, but it is a good plan to provide for the cleanliness of the buccal cavity by insisting on the thorough use twice daily of some simple antiseptic mouth- and throat-wash. Carbolic acid, listerine, sulphocarbolate of soda, or permanganate of potash, in suitable strength, will all be found useful for this purpose. Errors of digestion, which are frequent, must be corrected, and there is no condition of dyspepsia in which intestinal antiseptics play such a useful *rôle* as in migraine. Here, perhaps more than in any other disorder, soured milk, as introduced by Metchnikoff, finds its most useful application. In spite of the extravagant claims which have brought discredit on the lactic acid bacillus as an intestinal scavenger, there can be no doubt of its value in properly selected cases, and in one or other of its many varieties it will be found a helpful addition to drugs and diet. We are not concerned this afternoon with any detailed discussion of indigestion, except in so far as it is related to the subject of hemicrania, but it may be stated generally that, in the large majority of cases, its drug treatment in this connection resolves itself into the exhibition of acids or alkalies according to clinical indications, and that both groups of remedies are increased in their efficacy by the addition of some form of intestinal antiseptic. There are many combinations of remedies by which this plan of medication can be effected, and the two following prescriptions are merely quoted as examples which have, in my experience, been useful: (*a*) Dilute hydrochloric acid, ℥xv; pure carbolic acid, ℥ij; solution of strychnia, ℥v; chloroform water to 1 oz.; to be taken about one hour after each meal. (*b*) Carbonate of bismuth, 15 gr.; sulphocarbolate of soda, 10 gr.; tincture of nux vomica, ℥x; tincture of ginger, ℥xv; mucilage of acacia, *quantum sufficit*; peppermint water to 1 ounce; to be taken shortly before each meal. When the digestion, under the influence of one of these or other remedies, becomes comfortable, it is often necessary to provide for the permanence of the improvement which has been achieved by the administration of tonics. And since it is very frequently the case that anæmia is a factor in such cases, the value of a mild preparation of iron must not be overlooked. Such a prescription as the following, put up in capsule form, will, in many instances, prove efficacious: The citrate of iron and manganese, 4 gr.; Finkler's papain, 2 gr.; ichthyol, 3 gr.; extract of cannabis indica, $\frac{1}{3}$ gr., and extract of rhubarb, $\frac{1}{4}$ gr.; to be taken after meals, three times a day. When, instead of anæmia, the prevailing fault in the general health is nervous instability, the iron in the above capsule may be replaced advantageously by 3 grains of valerianate of zinc. In many instances cannabis indica is of special value; when this is ascertained to be so, the dose ought to be gradually increased up to the limits of the patient's tolerance. Quinine, arsenic, phosphorus, strychnine, and other tonics play a useful *rôle* in the treatment of many cases, and when it can be digested, cod-liver oil is of special value in feeding and toning up constitutional vigour. In cases which are accompanied by excessive arterial tension, the vaso-motor system seems to be balanced and rendered less liable to derangement by the regular administration over a continuous period of nitro-glycerine in combination with strychnine. The following formula will be found useful for such patients: Nitro-glycerine, $\frac{1}{100}$ gr.; strychnine, $\frac{1}{40}$ gr.; citrate of caffeine, 4 gr.; to be made into a pill and taken three times a day. Treatment on these lines must be continued, with intervals of rest from drugs, over a period of many months in order to secure a permanent improvement in metabolism and a durable condition of digestive competency.

In the treatment of the attack itself, the remedies which have been recommended are almost without number. It is sometimes possible to avert an attack by washing out the stomach with a weak solution of sodium bicarbonate whenever prodromal symptoms give intimation that an attack is

imminent. When the headache has developed there is nothing for it but to keep the patient closely confined to bed in a dark room, and to leave him, as far as possible, undisturbed. The severity of the pain is often diminished by the application to the whole length of the spine of a mustard plaster. It may also be relieved to some extent by keeping the head cool by cold water cloths or a simple evaporating lotion. Sedative liniments serve in some instances a useful purpose. Of the many local remedies that are advocated none are more efficacious than menthol or chloral, either of which may be used in such convenient combinations as the following: (*a*) Menthol and pure chloroform, of each one drachm in one ounce of soap liniment or olive oil; (*b*) chloral hydrate and camphor mixed together in equal parts. As regards drugs given internally, most good is achieved by the exhibition of one or other of the coal-tar derivatives. It is difficult to explain their *modus operandi*, but there can be no doubt of their usefulness. They are often most effective when combined with such other drugs as meet the idiosyncrasies of the patient or the special exigencies of his attack. In cases, for instance, where there is a high-tension pulse, the drug chosen to relieve the pain will act much more efficaciously if combined with one of the nitrites; in others, again, where tension is low and the general tone feeble, the "pain-killer" will do its duty twice as well if given in combination with an alcoholic stimulant, and so on. Antipyrin, phenacetin, antifebrin, pyramidon, and exalgin are among the most useful drugs for immediate relief, and they may be combined either with one another or with other remedies, according to requirements. Eventually every sufferer from migraine comes to know the prescription that affords him most speedy comfort, but it often requires many attempts before a satisfactory combination is found. In extreme cases, indeed, the pain will yield to nothing except a hypodermic injection of morphia, but in a disease so recurrent as migraine, the danger of creating a morphia-habit must never be lost sight of, and for this reason the drug should only be resorted to under the provocation of extreme necessity. The following prescriptions have often proved useful in my own practice: (*a*) Chloride of ammonium, 15 gr.; phenacetin, 10 gr.; codeia, $\frac{1}{3}$ gr. (to be dispensed either in the form of a powder or of two cachets). (*b*) antipyrin, 10 gr.; salicylate of soda, 10 gr.; tincture of gelseminum, ℥xv; spirits of chloroform, ℥xx; water to 1 oz. (*c*) Heroin, $\frac{1}{12}$ gr.; dilute hydrobromic acid, ℥xxx; citrate of caffeine, 5 gr.; compound tincture of cardamoms, ℥xx; chloroform water to 1 oz. In the case of all of them directions should be given for a dose to be taken every two hours for three doses; and thereafter every four hours until the pain is relieved.

In women, when the migrainous attacks recur only at the menstrual times, the use, for a week before each period, of a mixture of chloral hydrate, 10 gr., and bromide of sodium, 20 gr., given three times a day, is often successful in suppressing the attack or in diminishing its severity. When the temporal artery on the affected temple is prominent and throbbing, steady pressure over it may succeed in affording temporary relief.

July 25th, 1910.

General Anæsthesia with Part of the Circulation Shut Off.—Franke has applied the Esmarch bandage to the thighs or arms or both as a preliminary to chloroform anæsthesia, according to Klapp's suggestion, and has been impressed with the advantages of thus shutting off the blood in these regions from the influence of the anæsthetic, the anæsthesia being induced with much smaller amounts of the drug than with the ordinary technique. The method cannot be used in the case of varicose veins or eczema, joint and bone affections; it will evidently have to be restricted to healthy and vigorous limbs, but under these conditions he feels that it is destined to a wide sphere of usefulness, especially for correction of dislocations, fractures, incision of phlegmons and other brief operations. The patients passed under the influence of the chloroform without any phase of agitation, and it was rare at any stage of the anæsthesia. The breathing was always regular, and there was no cyanosis nor salivation; the pulse-rate increased a little after the band was applied and again when it was removed, in one case becoming a little irregular for ten or twenty minutes. There was no vomiting in any case, either before or after the narcosis, and the patients roused in less than ten or fifteen minutes after suspension of the drug. The return to the circulation of the blood overladen with carbon dioxide exerted a pronounced stimulus on the respiration centre, evidenced in each case by several deep breaths.—*Journ. A. M. A.*, vol. lv, No. 2.

SOME PRACTICAL POINTS IN THE MANAGEMENT OF DIFFICULT LABOUR.

A lecture delivered at the London Hospital.

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In some quarters it is the fashion to belittle the science and art of midwifery. We often hear something of this sort: "Labour is a natural process. Midwifery! What is there in it? The child is there; all that you have to do is to get it out. If it does not come by itself, pull it!" There is a good deal more in midwifery in civilised countries than this. If this were true, all that would be necessary for the equipment of a successful accoucheur would be a pair of forceps and a powerful frame. If you have got any such idea in your heads, and intend to practise midwifery, I beg you most earnestly to get it out again as soon as possible. Midwifery conducted on such lines means many stillborn infants, a high maternal mortality, and much impairment of health, sometimes permanent, in the more robust patients who survive.

Many of the difficulties that arise in labour may be avoided if a complete examination is made early in labour, or better still, before labour begins. The chief dangers in many cases of difficult labour arise from the fact that the accoucheur is confronted with the difficulty as an emergency: he has not foreseen it and laid his plans accordingly. Every woman pregnant for the first time ought to be examined long before the onset of labour, when she engages her doctor to attend her in her confinement. If she objects to be examined he should tell her that the object of the examination is to enable him to inform her whether the process of labour is likely to be straightforward, or whether any extra precautions will be necessary. He ought to be able to persuade her to understand that many of the "complications" of labour can be avoided or minimised if their presence is recognised in time. Most women know of some relative or friend who has had difficulty or danger in labour, and are grateful for anything that will encourage them about the straightforwardness of their own labour. Measurement of the interspinous, intercristal, and diagonal conjugate diameters will show if there is any severe degree of contraction of the pelvis. One is often unable to measure the diagonal conjugate in the normal pelvis of a woman pregnant for the first time if she is examined in the early months of her pregnancy, but one can at the same time recognise the fact that this failure is due to the firmness of the perinæal body and narrowness of the vaginal outlet, in other words that one cannot push one's fingers far enough upwards to feel the promontory of the sacrum, although they go up far enough to feel a promontory that was unduly low. If one cannot reach the promontory, and the interspinous and intercristal diameters are normal, one can safely assume that there is not much wrong with the pelvis. If the diameters of the false pelvis are small and the promontory cannot be reached, there may be general contraction. In such a case examination of the interior of the pelvis should be made later in pregnancy, when the soft parts have become more lax. It may be possible then to measure the diagonal conjugate diameter, and also to judge whether the pelvis is less roomy than it should be by the ease with which the lateral walls can be felt, and possibly by tracing an unusual extent of the brim by the finger-tips passed up from below. If there is any suspicion of slight contraction of the pelvis, the patient must be examined from time to time during the last eight weeks of pregnancy so that the accoucheur can determine whether the head can be pressed into the pelvis or not. Induction of premature labour at thirty-four to thirty-six weeks in cases of slight contraction of the pelvis gives excellent results in England, in spite of the unpopularity of this method of treatment in Germany and Austria. If the pelvis is too small to allow of the passage of a child at thirty-four weeks, the most satisfactory treatment will be to perform Cæsarean section at term. The urine of every pregnant woman should be examined for albumen. The trouble that a patient may be caused by having to send a bottle of urine to her doctor every two or three weeks is quite made up for by his assurance that it is all right. Few things cause more anxiety or more exhaustion to a doctor than an emergency call to a case of obstructed labour which he has to manage by himself, and looking at it for a moment entirely from his point of view, it is well worth his while to undertake the extra amount of work entailed by these preliminary

examinations if they will do away with some of these emergencies. Every patient, primigravida or multipara, should, if possible, be examined a few days before she expects her confinement. External version in a case of shoulder presentation is usually an easy matter before labour has begun; it may be very difficult or impossible during labour.

If you see the patient for the first time when she is in labour, make a systematic examination, abdominally and vaginally. Abdominal examination is sometimes unsatisfactory, but in many cases, particularly early in labour, it is much more valuable than vaginal examination. An occipito-posterior position cannot be diagnosed by vaginal examination early in labour, while at that time, when there is still plenty of liquor amnii in the uterus, its recognition by abdominal examination is usually easy. Again, the certain recognition of the face, the breech, or a shoulder at the brim of the pelvis early in labour may be impossible by vaginal examination; all that one can say is that the fœtal part occupying the pelvic brim does not feel like the vertex. Abdominal examination ought to make the diagnosis certain. Students should train themselves so that they always measure the diagonal conjugate diameter when making the first vaginal examination in labour. I have known maternity assistants diagnose correctly a slight degree of flattening of the pelvis during their first week on duty. If the patient has had previous labours her "obstetric history," *i. e.* the history of the character of those labours, is of great importance. Students are sometimes apt to neglect this. If she has borne good-sized children without difficulty her pelvis can be taken to be normal. If, on the other hand, she has had difficulty in any of her former labours, an attempt must be made to find out the nature and cause of that difficulty. The mere fact that one or more children were born with the aid of forceps does not of itself prove that there is contraction of the pelvis, but makes it necessary to investigate further. "How long was she in labour before the forceps were applied?" "Were any of the children still-born?" "Did her doctor say anything about the likelihood of trouble in subsequent labours?" etc. Some women of the hospital class intentionally withhold information unless they are definitely asked for it, *e. g.* a patient who has been told after a very difficult labour that she ought to have labour induced prematurely or possibly have an operation performed, may say nothing about this until she is in labour, "trusting to luck," and hoping that the doctor may detect nothing wrong. If there is any history pointing to contraction of the pelvis, an attempt must be made to push the head into the pelvis from above, and the pelvis must be measured. If it is found that there is contraction of a degree sufficient to cause much obstruction, a line of treatment must be decided on at once before either mother or child is suffering from the effects of obstructed labour. Cæsarean section or symphysiotomy must be performed *early* in labour; the risk to the mother is too great if her tissues are bruised and she is exhausted by prolonged labour.

One of the commonest causes of difficulty in labour is a persistent occipito-posterior position. When the vertex presents with the occiput behind there is a likelihood, especially in first labours, that the head will be incompletely flexed. Owing to the incomplete flexion, the forehead, which is in front, meets with the resistance of the pelvic floor first, and rotates to the free space under the pubic arch, and the occiput rotates into the hollow of the sacrum. If the head is of fair size it will require to be moulded considerably before it can be born, because, being incompletely flexed, the occipito-frontal diameter measuring four and a half inches is engaged, instead of the sub-occipito-frontal, which measures four inches. If forceps are used a very long and strong pull may be necessary. The forceps are likely to slip off, sometimes causing severe laceration of the posterior vaginal wall. I have seen almost complete rupture of the perinæum caused by ineffectual attempts at delivery of the head with forceps. If the head is delivered the child is not uncommonly dead, from prolonged pressure and injury by the forceps, and the mother is badly torn, complete rupture of the perinæum being caused by the passage of the unduly large diameter of the head. This is a very good example of rough-and-ready methods in delivery.

The diagnosis of occipito-posterior positions is not difficult as a rule. By abdominal examination the small parts of the child are felt all over the front, while the back is felt, not so distinctly, behind and to one side. By vaginal examination the anterior fontanelle is felt towards the front of the pelvis, and the posterior fontanelle towards the back. Sometimes there is so much œdematous swelling

of the scalp, and so much over-riding of the parietal over the frontal and occipital bones, that diagnosis by vaginal examination is difficult. In such a case the whole hand should be introduced into the vagina, if necessary under an anæsthetic, and then diagnosis will be found to be easy. Some men find the recognition of the occiput and nape of the neck easier if the left hand is used for internal examination. The finding of an ear is a great help in these cases, the free part of the ear pointing towards the occiput. Often the accoucheur knows how to recognise this cause of difficulty in labour without knowing how to remedy it. If he asks himself what has gone wrong in the mechanism of delivery, he cannot fail to see what is the proper treatment. What has gone wrong is this: the head has not rotated properly because it was not completely flexed. The ideal treatment is obvious—first flex the head and then rotate it. Push the forehead up as much as you can, and then grasp the head with your whole hand and rotate it, bringing the occiput to the front, helping the child's body round with your other hand on the anterior shoulder. This manœuvre is easy as a rule, particularly if the patient is under an anæsthetic. If you cannot give her an anæsthetic, you may not be able to get your whole hand into the vagina, but two fingers will probably be able to rotate the head, helped by the other hand on the anterior shoulder. It is not enough to rotate the head alone. If this is done the shoulders remain where they were, the neck is twisted, and when the hand is removed from the head the head rotates back again into its original position. When the occiput has been rotated to the front delivery may be left to nature, or the head may be delivered with forceps with ease and with no danger of severe laceration of the perinæum.

The fact that the forceps slip off the head or difficulty in locking the two blades should always lead the accoucheur to suspect that the occiput is behind, and make him investigate thoroughly.

If progress is slow during the second stage, although both pelvis and head seem to be of normal size, do not be in too great a hurry to put on forceps. There is a German saying that an English doctor always puts on forceps early because he wants to get home to a meal. A useful plan in a case where there is no obstruction and all is going well, except that progress is slow, is to fix a time and decide that you will not put on forceps before that time is reached, unless some new indication should arise. You will often find that before that time is reached labour is terminated spontaneously. Before delivery with forceps you should always make certain that the bladder is empty. A distended bladder is sometimes ruptured by delivery of the head with forceps.

A simple cause of obstruction is often seen in cases in which the membranes have ruptured too early, the anterior lip of the cervix being caught between the fœtal head and the symphysis, prevented from rising up, and becoming swollen with œdema. If the head is pulled past this obstruction with forceps the anterior lip of the cervix is crushed and may slough. It can be pushed up by two fingers in an interval between pains without difficulty, and will then be pulled up out of reach by the body of the uterus.

If you find slight contraction of the pelvis, make up your mind as early as possible whether you are going to deliver by forceps or version. Unless the contraction is only in the conjugate diameter and the head is presenting in some unfavourable way, version is not indicated. If the head is lying transversely, with the sagittal suture nearer to the promontory of the sacrum than to the symphysis, in a case of slight degree of flattening of the pelvis you cannot improve matters by version. Don't be in too great a hurry to put on forceps, but give the head time to be moulded. The fear of tonic contraction is to a certain extent a bogey. Tonic contraction will not come on very rapidly unless the uterus has been irritated by repeated manipulations. The rate of the fœtal heart-beat is an important factor in the decision as to the time when it is necessary to use forceps. If the fœtus is suffering from pressure the heart-beat becomes more slow. Presence of meconium in the liquor amnii in a case of head presentation is another sign that the fœtus is being pressed on to a dangerous extent. Although, as I said before, tonic contraction is not likely to set in until the second stage has lasted for a long time, the lower uterine segment becomes thin, even though there is a distinct interval between the pains. Application of forceps in such circumstances with the head still above the brim is a most dangerous procedure. In some of the Continental clinics application of high forceps by the assistants is absolutely forbidden. Not long

ago I had to open the abdomen of a patient and remove the fœtus and placenta, which were lying loose among the intestines, and then remove the ruptured uterus, ruptured by application of forceps to the head above the brim. The patient, fortunately, recovered, but she had been in great danger. Most cases of rupture of the uterus, one of the most terrible accidents in labour, are due to ill-advised attempts at delivery by version or forceps. The mortality which results from this accident is high, a mortality to be regretted all the more because the mother is often subjected to the greatest risk for the sake of a child that is already dead. The accoucheur has no right to subject the mother to the risk of version or forceps delivery in protracted labour unless he has made certain that the fœtus is alive. The conditions which must be present for version to be performed are: (1) A pelvis of sufficient size to allow delivery of the child through it. (2) Nothing approaching tonic contraction. There must be intervals between the pains, Bandl's ring must not be palpable, and there must be no perceptible thinning of the lower uterine segment. (3) If the second stage has already lasted long the child must be alive. If the head is above the brim and there are present signs of prolonged labour, the mother cannot be delivered safely except by embryotomy. In the rare event of the child being alive its life must be sacrificed. Cæsarean section, version, or high forceps might result in the birth of a living child, but the risk to the life of the mother would be too great. If there is any question of the life of the child *versus* the life of the mother, the child must be sacrificed. To quote Dr. Munro Kerr's words: "The mother may get another child, but the child cannot get another mother." It goes against one's instincts to perforate the head of a living fœtus in a case of protracted labour rather than to perform Cæsarean section, but it is the better treatment if there is any likelihood of the mother having been infected. I debated one night about four years ago as to which operation I should perform in a case where the patient had had forceps applied by two men before being brought to the hospital, and eventually decided, wrongly, in favour of Cæsarean section. The child did well, but the mother died on the tenth day of peritonitis. In a similar case in future in which a rise in the mother's temperature and pulse-rate pointed to infection, I should not hesitate to sacrifice the life of the child, repugnant though it would be to my instincts. If the head is in the pelvis and forceps fail to deliver it, or if it is above the brim in spite of prolonged labour, it should be perforated at once before the mother's tissues have been bruised by prolonged pulling, and before the lower uterine segment has been thinned to a dangerous degree. In some cases the accoucheur persists in attempts at forceps delivery from a sort of doggedness; he "doesn't like to be done." Doggedness is an admirable quality in its right place, but mulish stubbornness is apt to be mistaken for it in the treatment of difficult labour. Let the dogged man stop and consider what persistence in attempts at forceps delivery means. To the child it means risk of fatal compression of the head, risk of fracture of a cranial bone, and risk of intra-cranial hæmorrhage; to the mother it means risk of damage to the soft parts of the pelvis, resulting possibly in vesico-vaginal fistula, possibly in puerperal sepsis, and lastly, possibly in uterine rupture—all for the sake of a dead or damaged baby. Sometimes the accoucheur breaks one of the cardinal rules of midwifery, and after failing to deliver the child head-foremost, although the head has entered the pelvis, tries to push the head up out of the pelvis and deliver by version. Some men of large experience, gifted either with unusual clinical instinct or with an extraordinary amount of good fortune, succeed in delivering patients successfully in this way, but too often the result is what is to be expected, viz. death of the mother from rupture of the uterus. I have seen two cases of spontaneous rupture of the uterus, *i. e.* cases in which the rupture was not due to the action of the accoucheur, and at least a dozen in which the rupture was due either to forceps or to attempts at version. I shall have something to say about spontaneous rupture later. An inexperienced man may think that the absence of impaction means that the patient is not in any danger, but it is important to remember that absence of impaction may be due to the fact that the head has not entered the brim. If there have been good second-stage pains and the largest diameter is still above the brim, the case is just as urgent as if the head were impacted in the cavity of the pelvis. Careful examination in such a case will show that when the finger is passed up the anterior surface of the symphysis pubis the head can be felt to be bulging above

it, in other words the angle between the head and the symphysis opens downwards instead of upwards as it does normally when the head has entered the pelvis. In such a case there is nothing to be gained by delay. Attempts at version would probably cause rupture of the uterus; application of forceps would probably fail to deliver the head and would be very likely to rupture the uterus. If Cæsarean section is not to be performed immediately the head should be perforated at once. If the fœtal heart cannot be heard there is nothing to be thought of but immediate perforation. The head may be found to be impacted in the pelvis, as proved by the facts that the head does not move down with the pains, and cannot be pushed up between the pains. If impaction has been present for more than half an hour or so œdema of the vulva will appear—a most important sign when it appears for the first time during the second stage of labour. If the fœtal heart is audible an attempt should be made to deliver with forceps; if this fails or if the fœtal heart is inaudible the head should be perforated at once.

I want to impress on you as strongly as I can the need for protecting the vagina from laceration during extraction of the head after perforation, whether cephalotripsy, craniotomy, or cranioclasm be the method adopted. The vaginal walls are sometimes literally torn to ribbons during extraction of the head. They have very likely been bruised by ineffectual attempts at delivery with forceps and will tear very readily. Portions of the cranial bones as sharp as razors may project, and must be covered by folds of scalp or by the fingers during extraction of the head.

If the patient is a primigravida the head should not be dragged out of the vulva forcibly. When it has been moved well down it should be allowed to dilate the pelvic floor and vulval orifice during at least three or four pains, unless slowing of the fœtal heart-beats indicates immediate delivery. There is a tendency for the accoucheur, when he has overcome the obstruction, to deliver the head immediately and to forget that great care is necessary to prevent undue laceration of the soft parts.

Some of the above remarks apply to the treatment of transverse presentations. Before labour has begun and during the first few hours of labour it may be possible, and it is sometimes easy, to perform external version. If, when the patient is first seen, labour has advanced too far for this, the choice lies between bipolar version and internal version, the latter of which gives the child the better chance. It must not be attempted before full dilatation of the os. If the membranes rupture before the os is fully dilated the dilatation must be completed either by Champetier de Ribes' bag or by stretching with the fingers. The latter is a tedious and laborious process, but it must be persevered with until the os is really fully dilated. Difficulty in bringing down extended arms through the os when the child is not unduly large and the pelvis is not contracted proves that the os was not completely dilated before version was performed. Patience is just as necessary in manual dilatation of the cervix as it is in the management of labour with the breech in advance.

The simple plan of tying a noose of tape to a prolapsed hand, and, if necessary, tying another noose to the foot which is seized, should never be forgotten. If the membranes are ruptured and the os fully dilated when the patient is first seen a careful investigation must be made before internal version is attempted. The number of hours which have passed since rupture of the membranes is not the most important point. The fœtal heart must be listened for—absence of fœtal heart-sounds in a case of at all prolonged labour will at once indicate decapitation. If the fœtal heart-sounds can be heard, there are intervals between the pains, the ring of Bandl cannot be felt and the child is movable, and the mother's condition is good, the hand may be inserted cautiously through the os and the state of the lower uterine segment investigated. If it be not tightly stretched version may be performed; if it feel thin and tense the risk of causing uterine rupture is too great: the child must be decapitated. Fortunately decapitation of a living child is seldom, if ever, necessary. The operation of decapitation is an easy one and carries with it no special danger to the mother. The only difficulty lies in recognising the neck when the shoulder is tightly wedged in the brim. As soon as the neck is identified it can be cut through in less than a minute with Ramsbotham's sharp hook. The body is then extracted by pulling on an arm and the head is brought out by pulling with a finger in the mouth, aided by suprapubic pressure.

A pendulous abdomen, in which the uterus sags

forwards so much that the fundus points almost directly forwards, is a great source of danger in labour. Mal-presentations are common, but the patient is not safe even if the vertex presents. I saw in consultation a year or two ago a very stout multipara with pendulous abdomen whose uterus and vagina had ruptured a few hours after the onset of labour, long before there could be anything like tonic contraction. The head was presenting and the upper part of the vagina was filled with small intestine which came through a large rent in the posterior vaginal and uterine walls. In this case the fundus of the uterus probably pointed forwards and *downwards* at the beginning of labour. The posterior wall of the vagina and uterus lay on the outside of a curve, soon became stretched to bursting point and ruptured before any examination had been made, and before the patient had thought it worth while to send for her doctor. I sent her into hospital, opened the abdomen, removed the fœtus and placenta which lay loose in the abdomen, and stitched up the rent in uterus and vagina, draining through the lowest part of it, but the patient did not long survive the operation.

In cases of pendulous abdomen, as in many other complications of labour, prevention of difficulties is easier and safer than their treatment. The presentation should be ascertained, rectified if faulty, and then the uterus should be lifted up into its normal situation and kept there by a broad binder which takes the place of the abdominal muscles. If this is not done the patient, particularly if she is up on her feet during the first stage, runs the risk of a spontaneous rupture of the uterus, as in the case which I have just mentioned.

Difficulties met with in labour with the breech in advance.—If a large child is presenting by the breech in a primigravida with a normal pelvis, the best way to avoid difficulty is to perform external cephalic version before, or very soon after, the onset of labour. If the patient is not seen until labour has advanced too far for this to be possible, the accoucheur should do his best to ensure full dilatation of the os. If the membranes rupture before full dilatation a Champetier de Ribes' bag should be inserted. Bringing down extended arms in such a case may be a matter of extreme difficulty, and the presence of your forearm in the vagina, together with the body of the fœtus, is likely to cause some laceration of the perinæum. Patience during the birth of the breech and legs is absolutely essential; as long as things are going well, however slow the progress may be, you must not pull on the breech, because extension of the arms in such a case is more than an unfortunate occurrence—it is a calamity. The time seems very long, but you have to force yourself to inactivity. If the arms become extended in spite of all your care, you must pull the child down, holding it by the pelvic girdle, before you put up your hand to bring down the first arm. This pulling on the child may diminish the height to which your fingertips have to ascend by as much as three inches, and these three inches may make all the difference between life and death to the child. The wearing of rubber gloves is a great advantage here, the gloved hand slipping up more easily, with much less friction than the bare hand. The forceps should be ready for instant use in breech labour in a primigravida, if not in every breech case, in case jaw and shoulder traction fails to deliver the head.

Some patients clamour for chloroform early in labour, and consider that they ought not to be allowed to feel any pain at all. If the accoucheur accedes to their request the labour will be prolonged and tedious, the second stage pains poor, and there will very likely be trouble with the third stage, either undue delay in the birth of the placenta or hæmorrhage. If there is nothing, such as breech presentation or face presentation, to contra-indicate it, the patient should be encouraged to keep up during the first stage, walking about and sitting on a chair occasionally rather than lying in bed. She will probably tolerate the pain much better and will not be so anxious for chloroform. During the second stage she may have the acute edge taken off each pain by a few drops of chloroform—very few are quite sufficient—without any bad results.

In conclusion, let me urge you to study your cases when you are doing maternity duty. Experience is as valuable in midwifery as in any other branch of medicine, but some men acquire less of it from a hundred cases of labour than others acquire from twenty-five. It is better to see one case through than to attend half a dozen "B.B.A's."

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CONTENTS.

PAGE

*Skin Rashes and their Treatment. By J. L. BUNCH, M.D., D.Sc., M.R.C.P. 257

*A Clinical Lecture on Muco-membranous Colitis. By ARTHUR F. HERTZ, M.A., M.D., F.R.C.P. 263

*Aphasia and other Disorders of Speech. By TOM A. WILLIAMS, M.B., C.M. 268

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SKIN RASHES AND THEIR TREATMENT.*

By J. L. BUNCH, M.D., D.Sc., M.R.C.P.,

Physician in Charge of the Skin Department, Queen's Hospital for Children.

THE subject of rashes of the skin is a large one, and I do not propose to touch upon those common rashes which you are meeting every day, such as the rashes of the acute specific fevers, syphilis, etc., but rather to deal with a group which is not quite so well known or quite so easily diagnosed. I refer to the toxic erythemata.

There are perhaps few more troublesome and puzzling cases than those in which the practitioner is confronted with a scarlatiniform or morbilliform eruption, occurring in one or more individuals simultaneously in the same house or school, but where the rash is not quite typical. Some of these cases, of course, prove eventually to be genuine scarlatina or measles, but others do not. The apparently scarlatinal rash may be accompanied by sore throat, and the urine may seem to give a positive albumen reaction when tested with cold nitric acid, and the rash may ultimately desquamate, but the case is not one of scarlatina but of a toxic erythema. So, too, the morbilliform rash may closely simulate measles, not only in its characters, but also in its accompanying phenomena, but it is really due to a drug taken internally. Drug rashes have a wonderful facility for imitating well-recognised skin diseases due to other causes, such as urticaria, purpura, herpes, or scarlatina; they may take the form of macular or papular erythemata with a characteristically rapid peripheral spread, of vesicles, bullæ, pustules, nodules, or an œdema of inflammatory nature. The lesions are often polymorphous and may resemble erythema multiforme, or they may be complicated by the presence of

* A clinical lecture delivered at the Medical Graduates' College and Polyclinic.

some well-defined skin eruption due to an entirely different cause, as when, for instance, a copaiba rash co-exists with a secondary syphilide, or an arsenic eruption with a well-defined chronic eczema. Moreover, a drug rash will largely depend on the idiosyncrasy of the patient. In one individual the smallest quantity of belladonna or bromide will produce a well-marked rash, especially if he has kidney trouble, in another far larger doses of either drug will not bring about the slightest cutaneous lesion. Then, again, idiosyncrasy alone will account for the characters of the rash which is produced by a certain drug in a certain individual; thus, bromides may cause either acne, or nodular swellings, or a true bullous eruption or confluent furuncular lesions according to the predisposition of the patient. The reaction to a certain drug may greatly diminish if the drug is continued over a considerable period of time, or it may greatly increase and the patient's susceptibility become more and more marked, so that the smallest dose tends to produce a well-defined toxic erythema. Auto-intoxication may, apart from drugs, give rise to toxic rashes, and in such cases the poison is set free in the patient's own organism. Erythema of various kinds occurs in inflammation of the urinary and sexual organs, such as cystitis, pyelitis, nephritis and salpingitis. Similarly, when abscesses are present or long-standing unruptured buboes, angina, diphtheria, or various diseases of the stomach and intestine, in such cases the cutaneous lesions frequently take the form of erythema multiforme, or of urticaria. These rashes often occur also in the group of toxic erythemata *ab ingestis*, where the poisonous substance is tainted meat, fish, or tinned food. Allied to these are the serum rashes, morbilliform or scarlatiniform erythemata which are caused by the injection of diphtheria and other serums. In this connection we may also refer to enemata rashes, where absorption of toxic substances takes place from the large intestine after the injection of a soap-and-water or other enema; such rashes are frequently morbilliform.

The different forms of drug rashes and their causes can best be summarised by a classification such as the following:

(1) *Erythema* maculosum, papulatum, or figuratum.
- Quinine.
- Belladonna and atropine.
- Antipyrin.
- Copaiba balsam, cubebs, gonosan.
- Diphtheria and other antitoxins.
- Potassium and sodium iodides.
- Chloral hydrate.

(2) *Erythema* scarlatiniforme.
- Salicylic acid and salicylate of sodium.
- Quinine.
- Mercury preparations, especially calomel.

(3) *Urticaria*, with or without erythema.
- Antipyrin.
- Iodides and bromides.
- Salicylic acid and sodium salicylate.
- Santonin and valerian.
- Copaiba, cubebs, and turpentine.

(4) *Papulo-pustular* lesions.
- Iodides and bromides.
- Arsenic.

(5) *Vesicular and bullous* erythemata.
- Preparations of iodine and bromine.
- Copaiba balsam.
- Sulphonal.

(6) *Zoster.*
- Arsenic.

(7) *Purpura.*
- Quinine.
- Iodine and iodides.
- Chlorate of potassium.
- Salicylic acid and salicylates.

(8) *Nodular or furuncular* lesions.
- Iodides and bromides.

In this classification only the commoner drug eruptions are dealt with, from the point of view of the cutaneous lesions which most frequently result from the ingestion of certain drugs. Such a classification must necessarily be incomplete, as is shown by the investigations of Lewin, who examined 402 drugs as to their influence on the skin, and found that 204 (*i.e.* 50·7 per cent.) produced changes in the skin. Then, again, several forms of eruption often occur simultaneously in the same patient, or one form tends to pass into another. An erythematous eruption may, for instance, develop urticarial elements, and later on show bullæ or purpuric hæmorrhages. Arsenic is, perhaps, the drug which is most commonly given for long periods, and may at first only cause a pustular eruption, but, if persisted with, gives rise to well-marked pigmentation of the skin or epidermic thickening and hyperkeratosis. The spread of the eruption

far beyond the point of application is of course due to absorption, and this, too, accounts for the fever and general toxæmia, and the occasional involvement of the mucous membrane of the mouth and pharynx. In cases of iodoform poisoning, iodine can often be detected in the urine, and mercury in cases of mercurial toxæmia.

The *internal administration* of certain drugs frequently gives rise to rashes which are not easy of identification, especially in the early stages. The morphology of these rashes is characterised by a multiformity which, in practice, may cause the greatest confusion. They show themselves as localised or diffuse macular, scarlatiniform, circumscribed or universal erythemata, urticarial, vesicular or bullous eruptions, macular or papular rashes with peripheral growth and central involution, nodules resembling those of erythema nodosum, or purpuric hæmorrhages. The localisation of these rashes affords the greatest variety; sometimes they are symmetrical, sometimes asymmetrical; sometimes the mucous membranes are involved, sometimes not; their extent, the course they follow, and the combination of dissimilar cutaneous elements result in the production of a picture dissimilar to anything seen in the typical forms of skin disease.

The action of a drug given internally is most probably a direct action on the skin, being brought to the point of least resistance by the blood-stream and there producing its direct effect. But if this were so, we should expect the same drug, when applied directly to the skin from outside, to produce the same effect, and the fact that this does not always occur has given rise to the theory that during its absorption from the intestinal tract or its elimination, it gives rise to a toxin which is the active agent in producing the cutaneous lesions. This might perhaps explain cases such as that reported by Morel-Lavallée, where the occurrence of a drug eruption awakes such a predisposition in a patient that he then develops toxic erythemata from the ingestion of substances which previously had no harmful effect on him, such as strawberries, fish, wine, etc.

The external application of irritating chemical substances, if sufficiently strong, produces in most individuals definite cutaneous lesions. This is well seen in many trades where chemical irritants have to be handled over long periods of time. French polishers, for instance, who have to work with bichromate of potassium, dyers, tanners and painters who work with arsenic, lead and antimony, the workmen who have to do with chlorine, camphor and creasote, are all liable to be attacked by an acute or chronic dermatitis directly traceable to their occupation. This dermatitis may be papular, vesicular, pustular or exfoliative according to the reaction of the individual, and either trivial or severe. It is well known that some of the workmen who have to handle cinchona bark for the preparation of quinine are very liable to an irritating eczema, which involves the hands, arms and face on the slightest contact with the bark, while others are almost entirely immune. So, too, the vegetable dust in cotton, jute and hemp factories has an extraordinarily irritating effect on some persons and promptly gives rise to a dermatitis.

In the same way chemicals applied to the skin for medical reasons frequently cause a reaction entirely different to what was intended. Sulphur ointment is of course often given to cure scabies, but if it is applied thoroughly for more than three consecutive nights it frequently sets up a dermatitis which is hardly less irritating than the original scabies.

On the Continent, mercury is of course commonly rubbed in as a routine treatment of syphilis, and cutaneous eruptions are often the result. I do not say this in any disparagement of the inunction cure, for I believe this to be a most admirable method of treatment when properly carried out, but the possibility of mercurial rashes has to be taken into account, and these are usually of the character of folliculitis, erythema, eczema or purpura. Folliculitis frequent occurs when mercurial ointment is rubbed into a hairy portion of the skin: it first shows itself as a tiny nodule round the hair-follicle, which rapidly grows into a pustule. When blue ointment is rubbed in for the cure of pediculi pubis the same folliculitis often results, or the eruption may take the form of a morbilliform or scarlatiniform erythema, spreading even over the whole body, with intense itching or burning and sometimes a considerable rise of temperature. The inflammation subsides after a few days, the rash becomes paler and is succeeded by an exfoliation of large flakes of epidermis. The so-called mercurial eczema is a still more severe form of dermatitis, in which well-marked vesicles and blebs are associated with a diffuse redness and swelling of

the skin, and in rare cases it may go on to purpura. This disposition to mercurial eruptions may be either localised or general. Thus in the case of a girl, æt. 20 years, who was being treated for condylomata, the first inunction on the calves produced no effect, but twelve hours after the second inunction on the left forearm a dense, closely crowded erythema, having all the characters of an erythema multiforme, appeared on the flexor surface of the left arm. Treatment was omitted for a few days, during which the erythema disappeared, and did not reappear on the arm or elsewhere when inunction was again resumed.

It seems as if immunity to mercurial eruptions could sometimes be acquired.

Jadassohn, for instance, had a patient who developed a mercurial erythema after the application of calomel and grey plaster to the skin. Later he was given three centigrammes of tannate of mercury and a general erythema occurred as a result, but those portions of the skin to which the mercurial plaster had been applied and from which the previous rash started were the only portions of the skin which were not affected by the second erythema.

Mercurial lotions, gauzes impregnated with mercurial salts and applied to wounds, often set up a certain amount of dermatitis, especially when applied to places where the skin is thin and delicate, and of these the bicyanide of mercury and zinc is perhaps as little irritating as any. Chrysarobin and chrysophanic acid often give rise to a dermatitis, and here, again, those portions of the skin which have been affected with psoriasis and cured by chrysarobin may be the only portions of the skin which are immune from a subsequent chrysarobin dermatitis. Iodoform often sets up a dermatitis, in fact, practically all the usual chemical dressings, even boracic acid, may set up irritation in predisposed individuals, and the only surgical dressing which is absolutely innocuous is sterilised wool or gauze.

The inability to classify the cutaneous phenomena as belonging to any recognised skin affection should put the practitioner on his guard, and is frequently sufficient ground for the dermatologist to diagnose a drug eruption. The polymorphism of such an eruption may, therefore, be of great help in making a diagnosis, but it is found in practice that certain drugs administered to a peculiarly susceptible patient or given in excessive doses, tend to produce certain forms of skin eruption more frequently than others.

Arsenic eruptions of the acute variety generally take the form of a scarlatiniform or measles-like erythema. This usually involves the face, neck, shoulders and arms, is accompanied by œdema of the face and congestion of the conjunctiva, and may exceptionally extend to other parts of the body. For instance, a patient who had taken six grains of arsenious acid spread over a period of thirty-three days for furunculosis developed a gyrate erythema, with serious general symptoms, sleeplessness, irritating itching and burning of the skin, marked restlessness and excitement and intestinal symptoms. The erythema is followed by desquamation and cure, or it may be complicated by the appearance of vesicular elements. These are most frequent on the hands, but may involve the arms, feet, or scrotum. Bullæ may also supervene on the erythema, and give a picture resembling pemphigus. Cases have been recorded where varioliform lesions have followed the taking of arsenic internally.

Zoster does undoubtedly occur as a result of taking arsenic, and it may involve a spinal or cranial sensory nerve just as in an ordinary case of herpes zoster. Thus in 557 psoriaris patients who were treated with arsenic, zoster occurred in 2½ per cent., while in 220 psoriasis cases treated without arsenic not a single case of zoster occurred. When arsenic is given over long periods, keratosis of the hands and feet involving both palmar and plantar surfaces with marked callosities over the knuckles, but always symmetrical, may show itself; it has been seen after only four weeks' administration of arsenic. Epithelioma sometimes supervenes on the hyperkeratosis.

Melanosis, in the form of diffuse brownish patches, is a well-known result of prolonged arsenic administration, and I will only add that it is most marked in those areas which are normally pigmented. As mucous membranes are not involved in this pigmentation, a distinction from Addison's disease is thus established.

While the preceding skin eruptions are usually the sequelæ of arsenic taken for medical reasons, it must be remembered that arsenic can also be absorbed unwittingly. Wall-papers impregnated with arsenical colouring, dusting powders con-

taining arsenic, beer containing arsenic, sheep-washes containing arsenic, and numerous other substances have been the cause of severe arsenical poisoning, and the detection of the arsenic may only occur as the result of a correct diagnosis of the patient's symptoms.

Iodides, whether applied externally or taken internally, tend to give rise to an eruption which is more characteristic than the rashes produced by any other drugs. The commonest form is that of acne pustules, which involve the face, shoulders, chest and back, and are especially liable to affect individuals who have a natural predisposition to acne vulgaris. The lesions are reddish, with an erythematous base, surrounded by a pustule, varying in size from a millet-seed to a hazel-nut, projecting prominently above the level of the surrounding skin. The surface of the acne spot becomes scabbed over, and resembles either a variola pustule or an ecthyma pustule. On the face particularly these lesions may increase in size until they resemble large boils, of a dark coppery colour, or even anthrax nodules, flat or slightly pedunculated, with small sieve-like openings through which a quantity of pus can be squeezed. These excrescences run a painless chronic course, and leave smooth cicatrices. Such lesions may in their early stages be mistaken for those of erythema nodosum, or later on for gummata, or mycosis fungoides, and when mistaken for gummata the physician may be led to increase the dose of iodide, with most serious results to the patient. Audry has recorded a case which went on to gangrene. Eczematous eruptions are much rarer, but a circumscribed or confluent erythema, consisting of red or brownish patches involving the face and upper part of the trunk, is not so uncommon. The erythema may involve the whole of the body, like scarlatina, and in one case, after seven tablespoonfuls of a 3 per cent. potassium iodide solution, the erythema was followed by death.

Œdematous and pemphigoid eruptions due to iodides have also had fatal endings. The bullæ may be situated on healthy skin or may have an erythematous base, with contents which are either cloudy or blood-stained, and involve the face, scalp, chest, back, and fore-arms.

The mucous membranes of the mouth, nose, conjunctiva, and cornea may be the seats of such bullæ, and the resulting scarring has been known to produce such opacity of the cornea as to render the patient practically blind. Hæmorrhages complicate many of the iodide rashes, and a definite iodide purpura was first described by Fournier as affecting the anterior surfaces of the lower extremities, but leaving the knees and feet uninvolved. The purpuric spots are usually small, but may coalesce to form large patches, violet-blue or purple in colour.

These iodide rashes are usually due to the administration of iodide of potassium, simply because this is the commonest salt of iodine given internally It really seems, moreover, as if the potassium salt has some special action on the skin, as in the case of Besnier, where potassium iodide caused purpura of the lower extremities, but tincture of iodine given internally caused dyspnœa and rapidity of pulse, and no skin eruption. On the other hand, painting with tincture of iodine, or injecting it for hydrocele, has been followed by papular, pustular, and even bullous eruptions in other parts of the body. Iodoform may also cause a dermatitis.

Bromides produce rashes very similar to those of the iodides—acneiform, pustular, and furuncular, bullous, and purpuric. The occurrence of such rashes seems to be much more frequent, however, as a result of taking bromides ; in fact, it has been estimated that 75 per cent. of all patients who are treated with bromides develop a bromide eruption. It is certain that very minute doses are sufficient to produce an exanthem in certain individuals, and small doses tend to accumulate in the patient more than large. This is owing to the fact that large doses of bromides are diuretic, and one has frequently seen an eruption develop after the cessation of bromide administration, although none showed itself previously. Bromides are eliminated slowly, and three weeks after taking 15 gr. of bromide of potassium bromine can be detected in the urine and saliva, the maximum amount being present thirty-six hours after the dose is taken. A woman who had taken bromides for some time, but ceased taking them for a fortnight, suckled her child at the end of the fortnight, and the child developed a bromide rash. Bromides are absorbed rapidly by the mucous membranes, and can be detected in the saliva as little as three minutes after a dose. Bromide eruptions lead to difficulties of diagnosis similar to those of iodides, but I have already dealt with these.

Antipyrin rashes are the commonest of the rashes due to the synthetic febrifuges, but similar eruptions may also occur after phenacetin, salipyrin, lactophenin, etc. Such rashes are often of considerable extent and symmetrical, but this does not mean that they are always easy of diagnosis. I well remember one of the first skin cases to which I was called in consultation some years ago. The patient had a general morbilliform eruption, best marked on the extensor surfaces of the limbs, but presenting differences from any recognised skin disease. This in itself suggested a toxic erythema, and the patient readily acknowledged that she had been taking antipyrin off and on for some time, and had taken 10 gr. the day before the rash appeared. That the rash was due to antipyrin was proved by the appearance of a similar eruption on a second occasion when she took the same dose, but the puzzling factor in these cases is why the drug should at one time produce no skin lesions, at another time a well-marked general eruption in the same patient with the same dose. The most probable reason is that there is a temporary deficiency of elimination, by the skin, kidneys, or intestine, and this is borne out by the observations of Lewin, who finds that typhoid cases with constipation are especially liable to antipyrin rashes.

These rashes are usually irregular, hyperæmic patches on the trunk or limbs, or becoming general, morbilliform or scarlatiniform in character, and persisting for several days. The scarlatiniform rashes clear up with exfoliation of large flakes. When localised patches occur, these are especially liable to involve areas where skin and mucous membrane join, such as the lips, eyelids, and penis, and such patches often leave a brown pigmentation behind. Darker pigmentation still is seen on the penis, and Fournier described what he called "*verge noire*"—the skin of the penis becoming black as a result of taking antipyrin.

Urticaria, œdema and purpura also result from the administration of antipyrin, but of peculiar interest are those cases of bullous eruption accompanied by shivering and fever, which so closely resemble pemphigus that the diagnosis may easily be mistaken and the antipyrin continued, or the drug which is responsible for the mischief actually increased. The resemblance to pemphigus is increased when bullæ also occur on the mucous membrane of the cheeks and tongue.

Copaiba.—A patchy or confluent erythema may also result from the administration of cubebs, turpentine, or sandal oil, and when such a rash co-exists with a secondary syphilide the diagnosis is by no means easy. Otherwise the rash is likely to be mistaken for measles, scarlatina, or a syphilide, especially if there is fever and sore throat.

Quinine, morphia and chloral may all give rise to an erythematous eruption similar to the rashes already sufficiently described, but the fact that the eruption is atypical and there is a history of the administration of the drug will usually make the diagnosis clear.

Antitoxin rashes resemble either an erythema multiforme or urticaria, and are due to the serum injected and not to the antitoxin. They may be accompanied by arthritis, but in any case are not sufficiently severe to contra-indicate the use of, for instance, diphtheria antitoxin in a case of diphtheria. In other words, the benefits obtained from the antitoxin far outweigh the disadvantages of the eruption.

Treatment consists in reducing the dose, withdrawing the drug, or administering it in a different manner. Hypodermic or rectal administration will sometimes overcome the difficulty if it is absolutely necessary to continue the drug, and in the case of iodides and bromides the increased diuretic effect of large doses will sometimes cause a disappearance of the eruption which has been produced by small doses. But the sounder method is to withdraw the drug. In the majority of cases the eruption will begin to fade within a few days of discontinuing the drug and will soon disappear. The really important point, therefore, is the correct diagnosis of the toxic erythemata, and, however wide your experience, you will find that some of the diagnoses still present considerable difficulties.

August 1st, 1910.

Treatment of Paralysis of the Arm.—Helbing, in discussing the management of spastic cerebral hemiplegia and birth paralysis, emphasises the importance of over-stretching of the muscle as the cause of the trouble. When the ends of the muscle are brought closer together, correcting the over-stretching, the apparently paralysed muscle will often regain its function as he shows by some typical examples.—*Journ. A. M. A.*, vol. lv, No. 3.

A CLINICAL LECTURE

ON

MUCO-MEMBRANOUS COLITIS.*

Delivered at Guy's Hospital.

By ARTHUR F. HERTZ, M.A., M.D., F.R.C.P.,
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In neurotic individuals constipation is sometimes associated with the constant or intermittent passage of membranes of coagulated mucus and with attacks of pain, the condition being known as muco-membranous colitis.

Muco-membranous colitis occurs four or five times more frequently in women than in men. It generally begins between the ages of twenty and forty-five, but I have seen it in a child of three, and Dr. Goodhart tells me that in his experience it is by no means uncommon in children, as he has several times seen perfect mucous casts of the intestines passed by them.

Patients suffering from muco-membranous colitis are always neurotic, their abnormal mental condition dating from a period preceding the onset of the intestinal disorder. The disease is rare among hospital patients, the few cases I have seen in hospital being among brain workers, whose occupation involved a mental strain which was too great for them. In many cases actual neurasthenia or hysteria is present, and all the patients tend to become depressed and hypochondriacal. They invariably take a great interest in their condition and make very minute examinations of their excreta. The depressed condition of the nervous system in neurasthenia is generally associated with an abnormal irritability of the centres which influence visceral activity. This *faiblesse irritable* is the condition which more than any other predisposes to muco-membranous colitis.

A history of long-standing constipation is invariably obtainable. Diarrhœa may occur in the course of the disease owing to attacks of acute or subacute colitis, but in such cases the membranes disappear if the diarrhœa lasts more than a day or two. The constipation is largely nervous in origin, but it is often made worse by the insufficient quantity of stimulating food taken by the patient. It is constantly present, but is always most obstinate during the attacks of pain. It is then always associated with spasm of the colon, which is less intense and may even disappear in the intervals. Unformed mucus is often passed during the quiescent periods, showing that some catarrh of the colon is present, but large quantities of typical membranes only appear during the more definite attacks.

Attacks of muco-membranous colitis are often brought on by depressing emotions. They may occur at intervals of weeks or months, or may continue without intermission. The three cardinal symptoms of an attack are abdominal pain, constipation, and the passage of membranes, although I have several times known the latter to be passed without pain.

During an attack a dull aching pain or simply discomfort is more or less constantly present. Paroxysms of colicky pain may occur at intervals; they are occasionally accompanied by nausea and vomiting, and may be severe enough to cause the patient to faint. The pain is situated most frequently in the left flank and iliac fossa and just above the pubes; at the same time the descending, the iliac, and the pelvic colon are often tender and can be felt as a contracted cord, in which scybala can sometimes be distinguished. Less frequently there is pain and tenderness in the cæcum and ascending colon, which are felt to be more firmly contracted than usual. The passage of fæces and membranes, or of the latter alone, whether spontaneously or as the result of treatment, generally gives relief. In some cases, however, the discomfort is greatest for some hours after defæcation.

The constipation present in the intervals becomes much more severe during an attack. The stools consist of hard, small scybala, which may occasionally be moulded into thin or flat pieces as in spastic constipation; in many cases they are peculiarly pale in colour. Defæcation is often accompanied by painful straining, and it generally leaves a sensation that something is still present in

* The name *muco-membranous colitis* is adopted in preference to *mucous colitis*, because mucus is passed in excess in most cases of colitis, whether membranes are formed or not; in preference to *membranous colitis*, because there is a rare form of colitis, quite distinct from this in its ætiology, pathology, and course, in which fibrinous membranes are formed; in preference to *mucous colic*, because in many, if not in all cases, inflammation is present.

the rectum. On rare occasions a long period of constipation may be followed by spurious diarrhœa for several days, but stagnation of fæces in the colon is still present, as shown by the presence of fragments of hard fæces in the stools. The spurious diarrhœa is due to excessive irritation of the colon by the retained fæces leading to a localised acute or subacute colitis. The mucus is excreted as membranous shreds, which may in well-marked cases form tubular casts of the colon; these are occasionally so perfectly formed that the position of the follicles can be clearly recognised. In extreme cases the casts may be two or more feet in length; their diameter may be anything up to an inch and a half, and their walls may be exceedingly thin or as much as half an inch thick. They are often called skins by the patient and have been mistaken for tape-worms; they are sometimes rolled into a ball, which can only be disentangled by washing in water. The mucus is transparent like ordinary mucus or opaque like fibrin, for which it was formerly mistaken; its colour is grey-white, but it is often stained by fæces and rarely by blood. Occasionally solid cylinders or unformed masses of viscid mucus are passed instead of membranes.

The membranes may be passed alone or with scybala, and tubular membranes may enclose fæces. I have also seen large membranes embedded in a mass of hard fæces. Occasionally streaks or clots of blood are present, owing to hæmorrhage from excoriations of the mucous membrane or from anal ulcers or hæmorrhoids, all of which may be produced by the long-standing constipation and the passage of hard scybala.

Sir Andrew Clark was the first to prove that the membranes do not consist of fibrin, and subsequent investigations have shown that they are formed of coagulated mucin. The anomalous chemical behaviour of the membranes is due to the fact that the mucus is not merely dried or coagulated by acid, but has undergone a physical change, very similar to that which occurs in the conversion of fibrinogen into fibrin.

Microsopical examination shows that the membranes, which are otherwise structureless, contain a number of cells, most of which are in various stages of disintegration and obviously derived from the epithelial lining of the intestinal mucous membrane. Sometimes a small number of leucocytes are also present, and occasionally a few red corpuscles are found. The cells of all kinds are most abundant on the surface of the membranes and between the layers of coagulated mucin which constitute the membranes. Isolated nuclei are found embedded in the mucus, and many of the bacteria which normally inhabit the colon are naturally also present. In rare instances bacteria are present in excess owing to the primary condition being complicated by an infection with pathogenic organisms. In such cases the stools are generally very offensive, and attacks of pseudo-diarrhœa, which may be accompanied by pyrexia and symptoms pointing to a mild general infection, are likely to occur. The infection is generally superadded to the muco-membranous colitis, but in some cases the latter is a sequel of an attack of infective colitis, which may have been accompanied by severe diarrhœa. The infection may be secondary to a septic condition of the mouth or be due to contaminated food or water.

There cannot be much difficulty in diagnosis if the stools are seen, but a lady, sent to me on account of abdominal pain and constipation, had passed membranes for ten years without ever mentioning the fact to her medical advisers. In some cases membranes are only discovered on examining the ejecta after irrigation of the colon. The attacks of pain may closely simulate appendicitis or biliary colic, but the passage of the membranes clears up the diagnosis.

Associated conditions.—Muco-membranous colitis is occasionally secondary to the constipation which results from cancer of the intestine; it is therefore important to make an examination of the rectum and pelvic colon by the sigmoidoscope in all cases in which it is possible that cancer may be present.

The frequency of the association of appendicitis with muco-membranous colitis has been much exaggerated. Pain in the right iliac fossa is certainly a not uncommon occurrence in muco-membranous colitis, but in the majority of instances this is not due to appendicitis, but to the colitis affecting the cæcum and ascending colon. Some of the cases, in which the appendix is found to be normal after its removal for symptoms pointing to the occurrence of appendicitis, eventually prove to be muco-membranous colitis, the appendicectomy having produced no improvement. In very rare instances

chronic or recurrent appendicitis, by producing constipation, may give rise in predisposed individuals to muco-membranous colitis, which may then be cured by appendicectomy.

Nearly one half of the women who suffer from muco-membranous colitis have also some pelvic disease, such as uterine displacements, pelvic cellulitis, and endometritis. The association is due to the constipation, which results from the reflex inhibition of intestinal movements and which gives rise to the muco-membranous colitis. The spasm of the colon and the accompanying pain and tenderness are often increased during menstruation, probably as a result of alterations in the intestinal blood-supply.

Visceroptosis, including movable kidney, is frequently present with muco-membranous colitis. The association is not a result of kinking at the flexures of the colon, but is due mainly to the fact that the weakness of the abdominal muscles, which permits the viscera to drop, is a common cause of dyschezia. It is also probably due in part to reflex stimulation of the motor and secretory nerves of the intestines, produced by the drag of the dropped viscera on their sensory nerves. This view receives support from the benefit derived in cases of muco-membranous colitis associated with visceroptosis from temporary rest in the horizontal position.

Patients with muco-membranous colitis are frequently dyspeptic. There may be dilatation as well as ptosis of the stomach, this being due, like the constipation, to the depressed condition of the neuro-muscular mechanism of the alimentary canal, which often results from neurasthenia. Very little is eaten on account of the poor appetite, and because the patients avoid various articles of food, which they believe disagree with them. Hence they are pale and thin, and sometimes extremely emaciated.

The passage of *intestinal sand* is almost always associated with muco-membranous colitis. Its first appearance generally coincides with that of the membranes; occasionally it is only observed after the colitis has existed for several months or years, but it is probable that in many such cases its presence is at first overlooked. Only small quantities are generally passed, half an ounce being rarely exceeded. The gritty material resembles fine sand, and must be distinguished from the false intestinal sand formed of wood-cells, which is sometimes passed by individuals who have eaten a large quantity of pears, and more rarely of bananas. True intestinal sand is composed of pale yellow or reddish-brown granules, very irregular in shape, and never crystalline; they contain about 30 per cent. organic material, which consists largely of bacteria; the chief inorganic constituent is phosphate of lime. As intestinal sand is almost always associated with muco-membranous colitis, it occurs among neurotic and constipated individuals, and its passage is accompanied by severe colic. The mode of its production and its relation to muco-membranous colitis are quite unknown, but Cammidge has recently made the important observation that the quantity of inorganic ash is increased in all cases of colitis, whether an obvious deposit of sand is present or not.

Pathogenesis.—There has been much discussion as to the pathogenesis of muco-membranous colitis. As it is not a fatal disease, post-mortem evidence is scanty. A definite catarrh with an overgrowth of the mucus-producing cells has been observed in a number of autopsies, whilst in others the mucous membrane has been found to be perfectly normal, although even in these cases the rapid changes which occur in the intestinal mucous membrane after death would make it very difficult to exclude with certainty slight superficial lesions indicating catarrh.

The invariable presence of numerous epithelial cells in different stages of degeneration in the membranes shows that an abnormal degree of desquamation must be occurring from the mucous membrane of the colon. When, as is sometimes the case, leucocytes are also present, the evidence of some definite inflammatory reaction is conclusive. The part of the intestinal mucous membrane visible with the sigmoidoscope is sometimes healthy in appearance, but more frequently it is abnormally red and partially covered by mucus. In some cases temporary difficulty is experienced in introducing the instrument owing to spasm.

Nothnagel suggested that there are two different conditions, one nervous and one inflammatory, which he called respectively *colica mucosa* and *enteritis membranacea*. My own view is that mucous colic and mucous colitis are varieties of the same condition, and bear the same relation to each other that asthma does to asthma complicated with bronchitis. Two predisposing factors are invariably present—constipation,

and an abnormal nervous system. In the majority of cases the former is a result of the latter. Retention of fæces in an individual, predisposed by an abnormally irritable nervous system, may lead reflexly to local over-activity of the motor and secretory fibres of the colon, leading respectively to painful spasms and to excessive secretion of mucus. The supply of motor nerves to the intestines is well known; it has recently been shown that stimulation of the vagus in rabbits also leads to the expulsion of mucous casts of the intestines. X-ray observations have shown that serious delay in the passage of fæces rarely occurs before the hepatic flexure is reached, and in most cases it is confined to the part of the intestine beyond the splenic flexure. The seat of the spasm and of the production of excessive mucus probably corresponds with this, the small intestine being always exempt, and the more distal parts of the colon being more often affected than the proximal parts. I have, however, seen cases of muco-membranous colitis in which the delay was found by the X-rays to occur in the ascending colon; in these cases the pain and tenderness were confined to the right side of the abdomen.

If over-activity of the motor fibres occurs alone, enterospasm results. This is analogous to the production of pure spasmodic asthma by irritation of the bronchial mucous membrane by some peculiar atmospheric condition in an individual with an abnormally excitable nervous system. In other cases the spasm is associated with increased secretion of mucus, and "mucous colic" results; this is analogous to the more common cases of asthma, in which the bronchial spasm is accompanied by excessive secretion of mucus. The spasm in both cases causes retention of mucus. Roger has demonstrated the existence in the intestinal mucous membrane of a ferment—mucinase—which is capable of coagulating mucin in about ten hours. The retention of the mucus in mucous colic is of sufficient duration for this change to take place, and consequently a membrane forms ("muco-membranous colic"); the spasm in bronchial asthma causes the removal of the mucus to be so slow that the peculiar structures known as Curschmann's spirals form. In both muco-membranous colitis and asthma the expulsion of mucus is accompanied by relief of the symptoms. The irritation of retained fæces generally results in the production of a true catarrh ("muco-membranous colitis"), which is sometimes increased by injudicious treatment with irritating purgatives or injections; the severer the catarrh, the more active will the reflex be, which leads to spasm and secretion of mucus. A mixed condition of catarrh and nervous over-action is thus generally present, strictly analogous to the association of pure nervous asthma with bronchitis, produced by the action of the irritant primarily responsible for the asthma, or more frequently of the irritating fumes too often employed with the object of relieving the bronchial spasm.

The analogy between muco-membranous colitis and asthma is also seen in their ætiology. A similar type of individual is liable to the two conditions, both of which may sometimes occur in the same patient either alternately or simultaneously, or in different members of a family. Attacks of both muco-membranous colitis and asthma may be brought on by emotional disturbances and by trifling changes in external conditions.

Prognosis.—Muco-membranous colitis is a very chronic disease, and cases have been recorded in which it has lasted for thirty years. If treatment is begun at an early stage recovery generally follows, but when the condition has been present for many years the outlook is very unfavourable, and even if improvement occurs the likelihood of a relapse is considerable. The disease does not in itself shorten life, but when severe and of long duration it may cause a patient to become completely bedridden.

Treatment.—Patients who are, or have been, liable to attacks of muco-membranous colitis, should be treated with the object of removing the two underlying factors—the abnormal condition of the nervous system and the constipation. The patient should be encouraged to think as little as possible about his illness, and be forbidden to make minute daily examinations of his excreta. He should avoid overwork and excitement, but his mind should be kept well occupied, in order to leave as little time as possible for brooding over his illness and worrying about every trifling discomfort. A sufficient amount of exercise in the open air should be taken every day, but it is of the greatest importance to avoid undue fatigue. The patient should go to bed early, and in some cases it may be advisable to have breakfast in bed and only get up in the middle of the morning, but it is

generally unwise for the patient to be kept completely at rest, a Weir-Mitchell treatment being definitely contra-indicated. Removal from the home surroundings to a hospital or to some health resort is often very useful.

The constipation requires treatment by diet, massage, drugs, and enemata. In the majority of cases the best results are obtained with a generous mixed diet, containing porridge, green vegetables, fruit, and whole-meal bread, together with plenty of butter and cream, but irritation by skins and pips of fruit should be avoided, and in severer cases green vegetables should be passed through a sieve and prepared with butter. The food should be very well chewed, and all spices, mustard, pepper, and game should be prohibited, as they tend to increase the colitis. The thin and anæmic patient who has often unwisely restricted his diet should be encouraged to eat, even if anorexia be present; improvement in the appetite frequently occurs as soon as the weight begins to increase. A feeling of discomfort and fulness in the abdomen may occur at first, but it generally disappears within a few days of the change of diet. Sour milk is not specially indicated, as the colitis is secondary to constipation, and not due to an infection. When benefit has followed its use this must be ascribed to the stimulating action of the lactic acid on the bowels, and not to the presence of the active organisms. It would be equally effective if the latter were destroyed by boiling. I have on several occasions seen benefit follow gentle massage, applied particularly to the part of the colon, in which the X-rays had shown that stasis was most marked.

At the commencement of treatment every trace of fæces accumulated in the colon must be removed. This can best be done by the combined use of castor oil and high enemata; in severe cases injections of six to ten ounces of olive oil at night should also be given. Once the colon is empty, care must be taken to prevent the reaccumulation of fæces. In very mild cases a change in diet and the administration of belladonna are all that is required, but an aperient is generally needed at first in order to obtain a daily evacuation. The most effective is castor oil, taken regularly last thing at night or on waking in the morning. The dose must be regulated until one satisfactory stool is passed daily. As a rule, between half an ounce and an ounce is required. When castor oil cannot be taken a saline purgative should be used. The purgative is always most effective when combined with an anti-spasmodic, such as belladonna or codeine.

Mild cases can be completely relieved by general treatment, diet, and drugs, but most severe cases require local treatment in addition. This can be done at home, but it is generally more effective when given at one of the spas in which there are special facilities for intestinal lavage. Until recently Plombières and Châtel-Guyon stood alone in this connection, but since 1905 the same treatment has been very efficiently carried out in Harrogate. Between one and two pints of the water at a temperature of 100° F. are introduced into the colon through a long india-rubber tube from a douche-can, suspended at a height of one or two feet above the couch on which the patient lies. It is retained for ten or fifteen minutes, after which the patient gets up and evacuates the water, which is generally accompanied by some scybala and mucus. The proceeding is then repeated; a smaller quantity of fæces, but more membranes than after the first injection are generally expelled. The irrigation is followed by a bath at 100° F., and a douche at 110° F. is played through the cooler water of the bath on to the abdomen with a finely perforated nozzle. The treatment is continued daily for about three weeks.

There is probably no specific action in either the simple thermal water of Plombières or the alkaline sulphur water of Harrogate, the mechanical removal of fæces and mucus and the sedative action of the hot douche being the cause of the success of the treatment. Normal saline solution should be used if the treatment is given at home, and injections of soap and water, antiseptics, or astringents should be avoided, as they tend to aggravate the condition by irritating the mucous membrane. When the result is not satisfactory, olive oil may be injected in the evening and retained during the night before the high enema of water is given. Roger has found that bile contains an anti-mucinase which prevents the coagulation of mucin by mucinase; for this reason, ox-gall may be added to the oil. The effect of the intestinal douches is much increased if the bowels are opened naturally, or with the aid of a mild aperient shortly before they are given.

Various operations have been performed for intractable cases of muco-membranous colitis. In the very exceptional cases, in which all other treatment has failed, an appendicostomy, as advocated particularly by Keetley, should be made, so that the colon can be thoroughly flushed with water through the opening into the appendix and so kept empty. But the result has been unsatisfactory in many cases, and with the closure of the opening into the appendix there is a considerable probability of a recurrence, as the underlying nervous condition is unaffected.

During a severe attack the patient should remain in bed. The pain can generally be relieved by means of hot applications to the abdomen. Codeine and belladonna are the most useful drugs, and in very severe cases an opium suppository may have to be administered.

August 1st, 1910.

APHASIA AND OTHER DISORDERS OF SPEECH—DYSARTHRIA, APHEMIA, APRAXIA, IDIOGLOSSIA, AMNESIA, WITH CASES AND PROGNOSIS, DIAGNOSIS AND TREATMENT.

A Clinical Lecture before the Washington Medical Society.

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THE term "aphasia" is often so erroneously applied that it is not out of place to discuss some of the disorders of speech often thus misnamed: for there is the greatest therapeutic importance in knowing whether a speech disorder is (1) a permanent organic defect, congenital or acquired, or whether it is (2) a functional disability due to intoxication, œdema, or (3) arising purely from a vicious habit or fixed idea. A *resumé* of modern knowledge of this subject is the more useful in that the paper of Pièrre Marie (1), of Paris, gave to many the impression that the classical views of aphasia were false; and those who have not examined the question are unaware of the fact that the further studies (2) incited by Marie's article have established more firmly than ever the regional classification of aphasia.

It will be recollected that Marie denied the occurrance of *aphemia* in any form, by which term we denote the inability to utter words, although all the muscular movements for doing so can be managed when words are not in question. It is supposed that the *kinæsthetic* images are unavailable either by destruction of their centre, the left third frontal convolution, or by interruption of the path connecting this centre with the psycho-motor area concerned with moving the muscles of speech. Marie termed the loss of utterance *anarthria*, that is, absence of power to articulate, a most misleading application of a term always used in quite a different clinical sense; for it is applied only to cases which also show disability of the muscles to normally perform other acts as well as that of speech. This is clearly illustrated by the patient now shown (*anarthria*).

In her there was a gradual loss of clearness in the utterance of words and in the modulation of sounds; until now she is utterly unable to articulate a single word or to make any but the most elementary sound, in which it is easy to hear that the vocal cords are quite passive. She is truly anarthric, because, as you observe, her tongue is almost completely paralysed; the palate is highly paretic, as is the pharynx, for she is unable to swallow solids at all, and liquids are taken only with difficulty, and sometimes flow through the glottis. There is also a slight paresis of the lower facial and jaw muscles, with a tendency to dribbling of saliva. But she is not an aphasic: could the muscles move, she could utter the words she wants, of which she has a perfectly clear notion, just as she has of the act of swallowing, although she cannot perform it. Her disability is caused by the very rare condition, the partial necrosis of the homologous areas of the two hemispheres which bilaterally govern the movements of the tongue, larynx, pharynx, palate, and lower part of the face. So far as I know this case is unique, for no other areas are implicated.

Dysarthria is the condition heard in bulbar paralysis. It is there accompanied by atrophy of the muscles of articulation; but *pseudo-bulbar paralysis* resembles the case I have shown, for there is no muscular atrophy, but only an upper-neurone paralysis, arising, however, from infra-cortical lesions which are usually multiple, and proceed from arterial sclerosis.

The cases of *aphemia*, where the third frontal appeared intact, are explained by serial sections, which show that there is an interruption in the white fibres of the centrum ovale between F 3 and the temporal lobe (Mahaim, Congress of Geneva). The same observer reported two cases without motor aphasia in which the posterior two thirds of the insula were destroyed.

That the so-called lenticular zone of Marie has nothing to do with *aphemia* is shown by ten cases which Compte cut in series, in which the lenticular nucleus was free from necrosis, although small areas of destruction of the internal capsule had produced the dysarthria of pseudo-bulbar palsy.

When an aphasia is partial, it is usual for the substantives to disappear before verbs and adjectives, more especially in cases of cerebral tumours of the speech zone. The most likely explanation

of this is that most substantives are used less often than the common verbs and adjectives. Besides, it would seem by their use by primitive peoples that the verb and adjective are more deeply rooted in speech, as they are acquired first.

The inability to pronounce certain consonants or speaking in a nasal voice may be a congenital defect, or fault of training, but the nasal and thick voice quite often occur in the course of a *polyneuritis* from degeneration of the nerves to the palate. The prognosis in this case is quite good as a rule.

Idioglossia.—The most important element in the prognosis of *dysarticulosis of childhood* is the perseverance and intelligence of the mother and nurse. This is illustrated by a musician's child, æt. 4½ years, who was under my care in Paris on account of almost unintelligible utterance. I was not sure whether this was entirely due to some congenital defect of the nerve apparatus. Had I given up efforts after the mother had failed in two weeks to produce the least improvement, the former diagnosis might have been too quickly acquiesced in. But as I was fairly sure that training could overcome at least part of the disability, the father was urged to take the child in hand, and his efforts, seconded by those of the mother, completed a cure in four months.

We may pass now to the *treatment of motor aphasia.* The chief difficulty to conquer is the annoyance and loss of mental balance the patient feels in not being able to perform the habitual automatic action of speaking. Thus, *psychotherapy* becomes an important element of the training. A child is accustomed to being controlled and compelled to do things which he dislikes in the acquisition of arts. But it is very difficult for an adult to turn his energies into new activities. More especially is this the case with the difficult co-ordination required for a new automatic speech apparatus. One of the best methods is the learning of verse and its repetition. Very important is the utilisation of the motor images of the lips seen in a mirror, as in the training of deaf-mutes.

The better *prognosis* of aphasia as compared with aphemia is due to the constant re-education the patient's surroundings give. He is assailed all day long with words by hearing or sight, and has to make efforts to comprehend in order to understand his fellows. On the other hand, so difficult are the first words of the aphemic that inertia is apt to get the better of him, and efforts to speak cease to be made. As soon, however, as an improvement begins, each year will bring further gain.

Perhaps the greatest factor in prognosis is the intellectual and moral status of the individual. If the memory is gone, hope is small. If internal language has disappeared as well the prognosis is bad. Simple methods of testing the internal language are for the patient to indicate the number of syllables in the word representing an object shown him.

In the *diagnosis of aphemia* it is important—

(1) To exclude dysarthria.

(2) To exclude sensory aphasia, that is, word-deafness or word-blindness.

(3) To test the power of mimicry and the repetition of words. Spontaneous mimicry only disappears as intelligence is impaired.

(4) To test the singing, which is often retained along with the vocalisation, which in some people is merely an automatic accompaniment of the music.

(5) To test the reading power, which is nearly always at first slightly *alexic*, possibly through the mechanism which Von Monakow has termed *diaschisis.* This may be expressed simply as the interference with the function of one centre on account of the impairment of another centre with which the first is intimately connected physiologically.

(6) Writing with the left hand is sometimes possible; and it may be reversed as in a mirror; and the name can nearly always be signed, though rarely spoken in complete cases.

Differential diagnosis from sensory aphasia is easily effected by the transcription of letters, which is made in a servile fashion, and is really a drawing when there is word-blindness. *Simulation* is detected by its complete mutism; the real aphasic can always utter some sound; and it is characteristic that he makes most violent attempts to speak, while the simulator remains quite calm and makes no sound at all. The pretended mute's mimicry is not defective; and he has no difficulty of comprehension, and is not likely to show *apraxia*, unless he has been very well coached; besides, the true aphemic rarely attempts spon-

taneously to write with the contralateral hand, while a malingerer or hysteric readily does so.

A few words on *apraxia*, of which aphemia is merely a special kind. The term is used to denote an inability to perform an act conceived, and the substitution of some other act, an inability neither due to disorientation, amnesia, aprosexia (lack of attention), paralysis, ataxia, nor agnosia (inability to recognise objects felt, smelt, heard, and seen). It is very simply ascertained by demanding such acts as putting out the tongue, touching the ear, raising the leg, etc.; or by the less simple series of acts used in lighting a cigarette, cutting up one's food, and the still less simple acts used in dressing oneself, sealing a letter, etc.

The act of putting a cheque in the waste basket and pocketing the envelope is not strictly of the same order, for it is due to *failure of attention*.

Still another form is characterised by a general contraction of all the muscles of the limb concerned in the desired acts, which are less and less well performed as they are more complicated. The order of excellence in Kleist's case was (1) flexion and extension; (2) turning, sitting, arising; (3) going upstairs, standing on one foot, on tip-toe; (4) impossible to leap, to draw a circle in air with foot.

The faculty of performing orderly movements, *eupraxia*, is believed to reside in the left third and second convolutions. This centre guides the co-ordinate purposive movement complexes of limbs on both sides. Therefore the corpus callosum must be intact for its action. Its destruction does not seem to impair the *autokinetisms*, whether these are merely muscular *synergias*, such as (1) the extension of the wrist when the fingers are flexed; (2) the dorsi-flexion of the foot when the leg is lifted; (3) the bending of the knees when the trunk is extended backwards; or whether they are certain *habitual movements*, such as (4) walking, (5) shaking hands.

Sensory aphasia is really a form either of *amnesia* or of *agnosia*, and as a failure of recognition is in reality lack of power to remember either the thing itself, or to recall the connections which associate it with other things, amnesia becomes the chief factor in a sensory aphasia of the acquired type (*agnesia verbalis*). When congenital, moreover, it is the failure to fix the memories of words which gives rise to the word-blindness which perhaps occurs to a greater or less degree in about two per thousand school-children, who never acquire the power of reading with facility as a result of their school training. The powerful stimulus of some interest in after life, however, may call forth the extra effort needed for such defectives to learn to read freely. Thus one of Hinshelwood's cases (*British Medical Journal*, 1907, vol. ii) taught himself to read in four years through his interest in the football reports, over which he pored for four hours every evening in order to follow the game for which he had a passion. Seven years at school had hardly taught him to read a word.

Patients of this type can in reality always be taught to read if placed in a special class where the tactile images are cultivated by the use of blocked letters. It is believed that the defect is due to hereditary insufficiency of the associational area between the visual centre and the word-hearing centre. We do not know whether their training is made possible through the use of the right-side centre or whether it is not accomplished entirely by the association of kinæsthetic images with the sight of the written words, as in the deaf-mute.

The condition must be *diagnosed from ocular defects by* (1) the size of the letters making no difference to the reading power of the patient; (2) by the patient's ability to copy the letters; (3) by the fact that figures and some few words and letters are readily recognised. The general mental defect of *imbecility* is excluded by the fact that the child's auditory memory is quite good, and that all school tasks except reading are normal.

The same principles of diagnosis and educative treatment may be applied to *adult alexics*; but the prognosis is qualified by the facts mentioned when discussing aphemia, viz. the difficulty of a mature person to recommence the kind of education which every infant performs as a matter of course.

Word-deafness may be classified into three degrees. In the most grave, the patient does not know he is listening to speech at all when words are uttered in his presence. In the second, he does not recognise the language; and thirdly, in the slightest degree, he does not understand the meaning. The extent of this failure may vary from complete want of comprehension to an uncertainty of confusion only in complicated sentences.

The *prognosis* of the latter cases is fairly good

when the cause can be suppressed, for it would seem that considerable education of the auditory word-centre in the first temporal convolution is possible, and the patient can hardly help automatically listening to the words of his companions.

As the text-books contain clear and full descriptions of this affection I shall not enlarge, except to point out the distinctions between it and the *functional aphasias and amnesias of psychic origin.* The best way to do this is by the citation of cases recently seen.

The first is an example of aphasic amnesia of organic cause. It is introduced by way of contrast. It is characterised by the loss of power to express the thought in words, although the patient *feels* that the idea is there, and can sometimes explain it in a periphrasis. My patient, even after ten minutes' study, is unable to recall and relate in words the story of Noah Webster. The following is her account with much questioning :

It begins with N. *Noel?* No. *Noah?* No. *Newel?* No. *Noah?* That's like it. *Recognises Webster. College?* Yale. *Profession?* Lawyer. *What did he do when young?* Taught school. *What else?* Don't know. *A book?* Yes. *Kind?* Dont know. *Dictonary?* I suppose. *Date?* 1790; too late. *Why?* My idea earlier, but I don't get the rest. *Was it in war-time?* The other one. *Name? Against whom?* All other kinds in United States. *Civil?* Yes, that is it. *Date?* 1861. *Did Webster live then?* Too soon. *Against whom, then?* He was not old enough; don't remember now, though he taught school.

She did not recollect the capital of Germany, nor in what country Edinburgh was situated, although she had once been a school teacher; but she could reply quite intelligently to any question if she remembered the word to answer, and she could read and write; but in the latter there were frequent omissions of words, of which she was quite aware, and deplored it. She could not recall the word she wanted, although she at once recognised it when proffered. Her condition intermitted more or less, but never became normal. The importance of the case lay in its diagnosis from a purely psychic amnesia; and to ascertain whether it was not due to a chronic intoxication, or whether it was caused by the cerebral necrosis of arterial defect, as I finally decided, as metabolic treatment failed to ameliorate the condition, and its oscillations were very slight. That it was not a psychic case is shown by its entire unamenability to psycho-therapy, by its genesis apart from any psychological cause, and by the nature and extent of the responses to psychometric tests too long to detail here.

The second case, seen with Dr. Claytor at the Garfield Hospital, is an example which may be called *hysterical aphasia*, in which the *fixed idea* not to speak was a *reaction of defence* against the fear of relating some intensely humiliating experiences. Indeed, the patient tried to chase them from his thoughts, and the aphasia was partly in consequence of this.

Other psychic cases.—It is comparable to the reaction of one of Janet's patients, who, because she wished to conceal a fault from everyone, began to lie not only about that but about everything that she spoke. Another case of Janet's, on the contrary, became completely aphasic as a result of trying to conceal thought. The mechanism of a remarkable case described by Dr. Hickling is similar, but is derived from a fixed idea which is only part of a general paranoid trend. The patient becomes aphasic on account of the delusion that he has been ordered not to speak for so many hours by his hypnotiser.

Formerly the psychological mechanism of hysterical aphasia was a mystery; but modern *psycho-analysis* has taught us the why and wherefore (4). Case No. 2 is a very common type.

Another case which reacted in this way was conduced to by a metabolic defect which led also to *narcoleptic confusion.* Complete deafness and aphonia was the result of a most painful experience in a hospital in Idaho. The condition was recovered from quite suddenly by the stimulating touch of a detective.

Cerebral tumours as causes; faulty diagnoses.—*Hystero-neurasthenia* is quite commonly diagnosed to explain a paraphasia which later proves to be due to a cerebral tumour. Such was the case of the girl whom I observed in Paris, and who was treated as a neurotic and isolated for several months by so eminent a neurologist as Dejérine. In the autumn she entered the clinic of Ballet with a well-marked amnesic aphasia for substantives, very similar to that of the first case here reported. She had all along been inattentive and frivolous in her ways and had shown slight mental dulness. I watched the slow development of the papillœdema, which clinched the diagnosis, and a tumour was found in the left temporo-parietal sub-cortex.

Such recurring attacks of aphasia must be distinguished from those due to *ophthalmic migraine*; for both may be accompanied by headache and vomiting. The jargon-aphasia of ophthalmic migraine is, however, almost always accompanied by hemianopsia, and the attacks are of very short duration; and separated by a period of complete good health, whereas a careful examination of these tumour cases will reveal a defect of speech even between the exacerbations.

In conclusion, this sketch of a vast and complicated subject is designed to emphasise the extreme importance of early diagnosis of forms of aphasia which are purely psychic (5), and which are most amenable to proper treatment when taken early. Even in the organic forms early detection of the cause may enable us to impose means for its

suppression and thus to arrest, even though not to cure the patient's disease. Such are the cases of temporary aphasia which occur in the course of *arterio-sclerosis* through the variations of blood-pressure and osmosis in the weakened blood-vessels. The œdemas of *uræmic states* are of this variable type too.

Lastly, variable amnesia and slow comprehension may be one of the earliest signs of intra-cranial new growth, which may be revealed in an examination by one skilled in neurological technique (6).

I have said nothing of the defects of speech arising from faulty habits of mental attitudes such as stutterings, lalling, lisping, and the tics of speech, coprolalia, etc., in which the patient is compelled to utter obscene remarks, or barking, or coughing. These are psychasthenic phenomena, and form a chapter of neuro-pathology in themselves (7).

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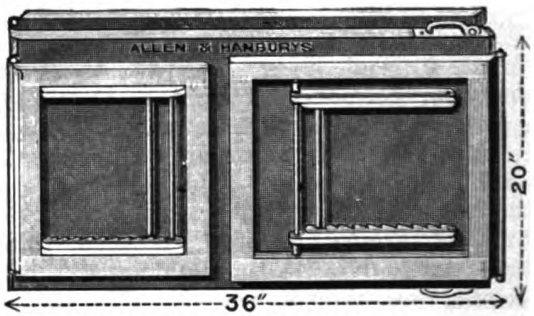
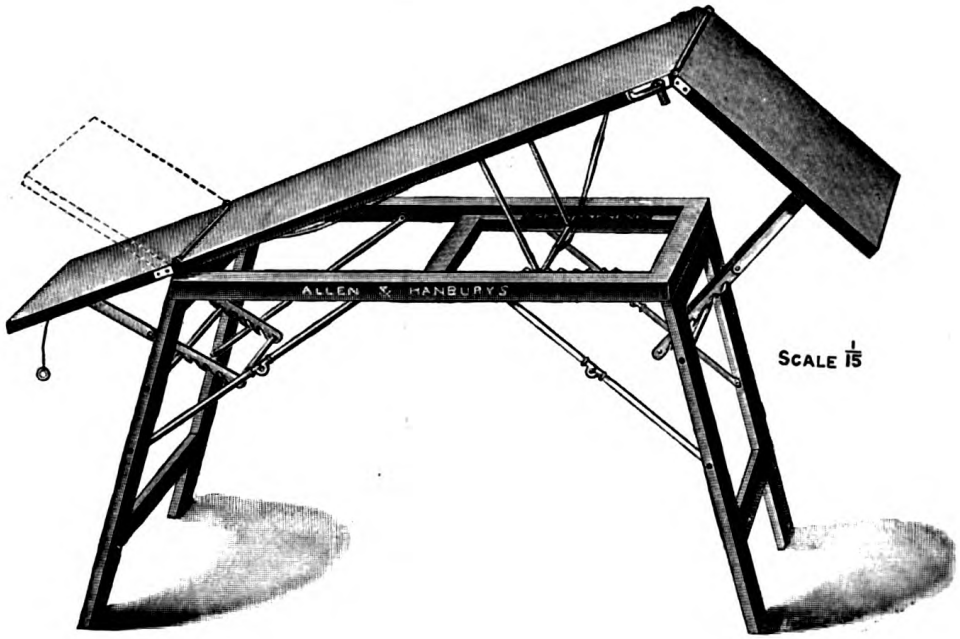
(7) Janet and Raymond, 'Les Obsesseûs et la Psychasthènie,' Paris, 1903.

August 1st, 1910.

Operating Table.—The accompanying illustrations show a portable form of operating table. It is light, weighing only about 40 lb. complete, very portable, measuring when folded up 36 in. by 20 in. by 5 in., so that it can quite easily be placed inside or on the top of a motor-car or carriage, and very strong, being manufactured from oak varnished with hard varnish, and steel, nickel plated; all joints and working parts are made of metal. It is adjustable to almost any position, and that portion of the table which supports the patient's legs is made to adjust to any angle in order to reduce the pressure on the popliteal spaces to a minimum. The head part of the table can be very easily raised or lowered. It is a useful table for surgical or gynæcological operations and clinical examinations, especially when the administration of an anæsthetic is necessary, or when it is wished to use a cystoscope, urethroscope, sigmoidoscope, œsophagoscope, or bronchoscope. Messrs. Allen & Hanburys, Ltd., are the makers.





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CONTENTS.

PAGE

*The Non-surgical Treatment of Ulcer of the Duodenum. By GEORGE HERSCHELL, M.D. 273

*A Clinical Lecture on Retention of Urine. By HUGH LETT, M.B., F.R.C.S. 281

*On some Pitfalls in the Diagnosis of Common Diseases of the Eye. By A. HUGH THOMPSON, M.A., M.D., B.C. 286

* *Specially reported for the Clinical Journal. Revised by the Author.*

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THE NON-SURGICAL TREATMENT OF ULCER OF THE DUODENUM.

By GEORGE HERSCHELL, M.D.Lond.

I AM convinced that the chief reason for the want of success so frequently experienced by those attempting to cure a duodenal ulcer by medical means is the half-hearted manner in which the treatment is carried out and the neglect to make use of the results of recent scientific work. If the alleged failures are investigated, it will be found that in most of them the patient was simply put to bed and placed upon a milk diet, with or without a preliminary period of rectal feeding, with, naturally, failure as the result. As I hope to presently show, we have many more resources at our disposal, and if we will only take the trouble to use them our failures will be limited to the comparatively few cases in which either an extensive thickening or adhesion offers a mechanical obstacle to the healing process or in which a stenosis or hæmorrhage demands surgical interference.

When we have established to our satisfaction the presence of a duodenal ulcer, the question will at once confront us as to whether the case should be handed over to the surgeon, or whether we are justified in attempting to cure it by non-surgical means. The answer to this will depend upon whether there is any marked interference to the passage of food along the duodenum, or immediate danger to life from hæmorrhage, or perforation, actual or impending. In these conditions surgical measures are urgently demanded. In their absence we may, I think, undertake the medical treatment of a duodenal ulcer with a distinct probability of success. There is, however, another factor which should weigh with us in making our decision. It is whether the patient will give us a fair chance of curing him by medical means. It is quite useless to attempt to cure a case of duodenal ulcer unless the patient will submit to the very monotonous and

insipid diet for a sufficient length of time, and is prepared to spend two or three weeks in bed.

To treat with success a duodenal ulcer it is necessary to have first of all a clear idea of the rationale of the methods we are making use of, and to that end we must devote some study to what we know of its causation.

The tendency of modern research is to show that a duodenal ulcer is caused by the direct action upon the intestinal wall of trypsin and other proteolytic ferments contained in the digestive fluids, and set free from disintegrated tissue-cells and leucocytes. In a condition of health the cells forming the duodenal wall are protected against these ferments by the presence of anti-enzymes and anti-lysins in the blood-serum. Under certain conditions at present not perfectly understood these anti-bodies are absent from the blood-serum or present in diminished amount, and in consequence the duodenum becomes vulnerable to the lytic fluids with which it comes in contact. A local lesion is in consequence liable to be set up, especially at the spot upon which impinges the jet of chyme from the stomach, or at a point of local stress such as an extravasation, or the drag of a gastroptosis upon an adhesion.

The cause of the diminution of anti-bodies in the blood-serum has not yet been worked out. Whilst on the one hand it is of course possible that it may be, as Dr. Hort suggests, "a symptom of a general disease as yet unidentified,"* I think that it is much more likely to be the result of the health-deteriorating conditions with which it is so often found associated in practice. These are mainly—

(*a*) *Pyorrhœa alveolaris.*—Quite a number of sufferers from relapsing duodenal ulcer have bad pyorrhœa, and it is easy to understand that the continuous daily introduction of large amounts of toxins into the body must exercise a degrading influence upon the blood. It is not impossible that virulent organisms swallowed with the food may actually assist to set up the local mischief, especially in patients in whom there is deficiency in the amount of HCl in the gastric juice (achylia gastrica). Under these circumstances the organisms will pass in full vigour through a stomach which has been deprived of its natural protection.

The especially virulent organism appears to be a spirillum which is Gram-negative, and I have on so many occasions noticed its presence in extremely large numbers in the pyorrhœal pus of patients with duodenal ulcer that I feel the coincidence may be more than accidental.

(*b*) *Auto-intoxication from the intestinal tract.*—Sufficient attention has, I think, not been paid to the experiments of Turck, of Chicago, who found that he could produce gastric ulcer at will by feeding dogs with living colon bacilli, the positive results being 100 per cent. with no failures. The blood of all the dogs was sterile, showing that the result was due to a toxæmia and not to the actual presence of bacteria in the blood. The inherent probability of this was confirmed by the observations of Rosenau and Anderson, of the hygienic laboratory of Washington, who noticed that among the guinea-pigs used to standardise diphtheria antitoxin there were areas of congestive hæmorrhage and ulceration at or near the pylorus in at least half of those which died before the tenth day, proving that it was possible for a toxæmia to set up such local lesion.

There are at present unfortunately no statistics available as to the concurrence of duodenal ulcer and intestinal putrefaction, as the routine examination of the stools has been unaccountably neglected in this country. My own observations, shortly to be published, tend to show that in the majority of cases which have come under my notice there has been a marked amount of intestinal putrefaction, the signs upon which I have relied being the following:

Relative excess of Gram-positive organisms, especially large forms, in stained slides of the stools, alkaline reaction of the stools, relative excess of anaërobes, excess of indican in the urine, and increase in the co-efficients of Combe and Amann and in the capillary constant. It is a noteworthy fact that the great increase in the number of cases of duodenal ulcer (and also of appendicitis) met with of late years coincides in point of time with the development of methods of cold storage and the consequent consumption by the masses of meat and poultry killed many months before. I am sure that in eating many birds which have been thus preserved in cold storage since the previous year we are unconsciously repeating upon ourselves the experiments of Turck.

(*c*) *Anæmia.*—In many cases anæmia is met with

* 'Brit. Med. Journ.,' 1910, vol. i, p. 76.

in conjunction with ulcer of the duodenum and whilst we have as yet no knowledge of any relationship between the two, the important work of Fütterer* is worthy of note. By a series of experiments upon animals he demonstrated that an ulcer made by excising a small portion of the mucous membrane of the stomach could be prevented for an indefinite period from healing if the animal was kept in a condition of anæmia by the administration of hæmolytic substances.

Taking all these facts into consideration it is obvious that the scientific treatment of duodenal ulcer should be conducted upon the following lines:

A. *The Elimination of any Condition which may possibly account for the Deficiency of Protecting Bodies in the Blood.*

With this end in view, as soon as we commence active treatment (1) a thorough examination must be made of the condition of the teeth and gums.

This should be preferably carried out by a dental surgeon, as although in many cases pyorrhœa alveolaris may be so far advanced as to be obvious on the most cursory examination, in others it may be so limited in extent as to be diagnosed only by the expert observer. In mild cases pyorrhœa may be cured by scaling and the application of drugs such as nitrate of silver, aromatic sulphuric acid, or iodine, to the interior of the pus-pockets, together with the employment of a vaccine prepared from the organisms present. When at all severe the affected teeth should be extracted, for the reason that under the most favourable circumstances the cure will be lengthy, and quite apart from the pus which is swallowed, during the whole of the time toxins will be continually absorbed from the pockets into the blood.

(2) A Wassermann test should be performed in all cases in which there is the slightest possibility of there being a specific infection.

This test has now been so simplified that it can be carried out by any practitioner without a laboratory and without previous experience.† If the test is positive the prognosis is good, as a rapid cure will probably follow appropriate and energetic treatment.

(3) The intestine must be disinfected in all cases in which there is evidence of abnormal putrefaction.

In the light of our present knowledge the best method of accomplishing this is by the administration of the Bulgarian bacillus (bacillus of Massol). As there is an ulcerated surface in the duodenum which we do not wish to irritate we must not give it in the form of soured milk or any other liquid culture which will contain lactic acid. We must make use of a dried preparation, of which there are several on the market quite reliable if used within a few weeks of manufacture.* The tablet should be crushed to powder and given in suspension in sweetened water, on an empty stomach. The bacilli will then pass at once through the stomach and probably arrive in the large intestine before they regain their activity. In this manner there will be no danger of irritating the ulcer. The particular tablet which it is proposed to use should be carefully tested in the following manner to demonstrate whether it contains living bacilli. It should be crushed up in milk and placed in an incubator for a few hours. An agar plate should be inoculated with this in the usual manner and incubated. If the characteristic colonies of the Bulgarian bacilli do not develop the sample should be rejected and trial made with another brand.

In cases in which the putrefactive changes are evidently mainly in the large intestine much benefit will be derived from high injections preferably given in the form of continuous irrigation by means of a double tube. I do not advocate the use of any disinfecting drug for this purpose, as the bowel can be cleansed with perfect efficiency with normal saline solution.

(4) Anæmia must be treated.

In treating cases of duodenal ulcer with iron preference should be given to the hypodermic method, because not only do iron salts frequently irritate the stomach and intestines, but they will obscure the reaction for occult blood in the stools and thus deprive us of a valuable method of watching the progress of the case. The preparation of iron which I use is the citrate, which should be in aqueous solution properly sterilised, each dose

* 'Festschrift f. Rindfleisch,' 1907, p. 89.

† "A New and Simple Method of Performing Wassermann's Test," by R. Weiss, 'Med. Press and Circular,' June 22nd, 1910, p. 637. [A case containing the complete apparatus required is sold by the Roborat Company, 8, Harp Lane, E.C.]

* *Vide* 'Soured Milk and Pure Cultures of Lactic Acid Bacilli in the Treatment of Disease,' Herschell, London, 1909, 2nd edit.

being put up in hermetically sealed bulbs as in the case of vaccines. The daily dose may vary from a quarter to three quarters of a grain. This method of administering iron has the endorsement of Peters* and Morse,† who have used it extensively in this manner. When indicated by the condition of the blood the iron may be combined with arseniate of soda.

B. *An Attempt to Remedy the Deficiency of Anti-tryptic and Anti-lytic Substances in the Blood.*

The presence of anti-enzymes and anti-lysins in normal serum having been definitely established by laboratory work, its empirical administration as a possible method of re-establishing the natural condition of immunity of the gastric and duodenal mucous membrane was first suggested by Dr. Hort in 1907. On the whole the clinical results obtained by it have been surprisingly good, and more than justify its routine administration. Normal horse-serum has the advantage of not only containing a very large quantity of anti-bodies, but also of being an article of commerce and easily obtainable.‡ In cases of duodenal ulcer it is best given by the mouth because we avoid the risk of the unpleasant toxic effects occasionally following the hypodermic administration of serums, because we are able to avail ourselves of its direct stimulating effect upon the surface of the ulcer itself, and for the reason that it will fix any hydrochloric acid and residue of tryptic and peptic bodies remaining uncombined with the proteid of the food in the stomach. For the last reason it should be given directly after food. I am sure that some of the comparatively unsatisfactory results which I obtained in the first cases which I treated with it were due to the fact that I administered it when the stomach was empty. The direct healing action of the serum upon the ulcer is probably of considerable importance. Serum has been shown to contain not only the protective bodies which antagonise the destructive toxins of cells and bacteria, but also substances which stimulate the repair of tissues. These *stimulins* can be demonstrated by the reaction produced when normal serum is applied to an old sluggish superficial ulcer. Hort, in fact, further suggests * that each batch of horse-serum should be tested in this manner for its stimulin content before being used therapeutically, and rejected if no marked reaction is produced in thirty-six hours. Horse-serum is supplied in glass containers each holding a single dose of 10 c.c. Latterly it has been found that it is possible to considerably increase the anti-lytic properties of normal serum by special treatment, and it is probable that serum modified in this manner will be used in the cure of duodenal ulcer to the exclusion of the ordinary serum of commerce.

C. *The Reduction in the Acidity and Peptic Power of the Gastric Juice.*

This may be accomplished either by acting upon the gastric juice after it has been secreted, or by taking measures to inhibit its secretion. We may reduce the activity of the secreted gastric juice by neutralising its contained acid by means of alkalies, fixing it by the inclusion in the diet of substances having a high combining power, or by the administration of articles of food which tend to inhibit the action of the peptic ferment.

It was formerly supposed that an excess in hydrochloric acid acidity in the gastric juice was an important factor in the production of gastric and duodenal ulcer. We know now that such is probably not the case, and that granted a reduction in the resisting power of the gastric or duodenal mucosa, gastric juice of normal acidity is quite capable of determining an ulcerative process. That the reduction in the acidity of the gastric juice is an important factor in the cure of duodenal ulcer must be apparent from the experiments of Bolton, who found that the gastric ulcers which are invariably caused by the injection of gastro-toxic substances into the peritoneums of guinea-pigs were not formed if the acidity of the gastric juice was kept below normal by neutralisation.

Alkalies not only neutralise the acid actually present in the stomach, but in repeated doses go far towards depressing the energy of the secreting cells, so that a diminution in the output of hydrochloric acid will continue for some time after their administration has been discontinued.

It is important to bear in mind that in practice all alkaline drugs cannot be used with equal advantage, as they have different combining powers, and

* 'Med. Rec.,' October 10th, 1908.

† 'Journ. of the Amer. Med. Assoc.,' July 10th, 1909.

‡ Normal horse serum, prepared by the Lister Institute, Allen and Hanburys; and Burroughs Wellcome, Snow Hill.

* 'Brit. Med. Journ.,' 1908, vol. ii, p. 1081.

the products of their combination vary in irritating properties. Bicarbonate of soda combines with about half its weight of hydrochloric acid, and produces as the result of the decomposition carbonic acid and sodium chloride. Magnesia usta combines with twice its weight of hydrochloric acid, producing magnesium chloride. The result of the action of calcium carbonate upon hydrochloric acid is calcium chloride, which is far more irritating to the stomach than the salts of sodium and magnesium. Magnesium salts tend to relax the bowels and those of calcium constipate. Bicarbonate of sodium has the disadvantage that it is immediately soluble, and consequently the whole of the alkali administered is thrown into the fluids of the body. With magnesium and calcium salts only sufficient is decomposed to neutralise the acid actually present. Carbonates and bi-carbonates disengage carbonic acid gas, which tends to produce flatulent distension, whilst magnesia usta is free from these drawbacks.

The dose of alkalies required may be estimated from the fact that according to Bidder and Schmidt the human stomach secretes about 500 c.c. of gastric juice per hour during the digestive period. Recognising at least ten hours of active digestion a day we arrive at a daily excretion of five litres. Estimating the HCl content of this at 1·02 per cent. we shall have a daily average secretion of ten grammes of pure HCl. To neutralise this we shall require twenty grammes of bicarbonate of soda, or five grammes of magnesia usta. In actual practice we shall not require anything like this quantity, as we shall have to take into consideration the amount neutralised by the proteids of the food. A word may be said about lime-water, which has been added to milk for at least a hundred years with the idea of acting as an antacid and neutralising the excess of acid in the gastric juice. When one takes into consideration the fact that the aqua calcis of the British Pharmacopœia only contains 1·28 parts of lime per litre, we can see that from nine to ten litres would require to be consumed to neutralise the hydrochloric acid secreted in one day. It is more probable that the undoubted action of the lime-water in furthering the digestion of milk is due to the calcium chloride, which assists the rennin to form insoluble casein from the caseinogen of the milk.

The action of bismuth appears to be mainly to act as a protective to the surface of the ulcer, and according to some authorities to increase the secretion of mucus.

With these facts before us we can deduce certain rules for the use of the alkalies in the treatment of gastric and duodenal ulcer.

(*a*) The best results will be obtained by combining the quickly soluble bicarbonate of soda with one of the more insoluble salts.

(*b*) We should combine bicarbonate of soda with magnesia usta when there is a tendency to constipation, and with calcium carbonate when there is looseness of the bowels. And when giving magnesia we should replace it with calcium carbonate if at any time the bowels become loose.

(*c*) We may combine a small quantity of bismuth with the other drugs with the object of protecting the surface of the ulcer.

(*d*) We may add lime-water to milk, not with the idea of neutralising much of the HCl of the gastric juice, but to assist the digestion of the milk.

There are three other points which must be considered in prescribing alkalies:

The excipient to be used.—It was formerly the practice to prescribe bismuth, magnesia and similar insoluble powders, together with mucilage of tragacanth or acacia "to swing" or suspend them. The disadvantages are that not only is there a liability for the powder to cake at the bottom of the bottle, but the drug used is to a certain extent protected from the immediate action of the acid present and does not neutralise it so promptly. The consensus of modern opinion is that alkaline powders are best prescribed either in cachets, or in powder form to be stirred up in water at the time of taking.

Palatability.—This is a practical point which should not be neglected. All medicine is disagreeable enough without having it more nauseous than necessary. When prescribed in powder form we may add with advantage a little sugar of milk, saccharin, or an aromatic spirit such as peppermint, which will be taken up well by it.

The addition of belladonna or atropine.—As we shall presently see, these drugs have quite an extensive use in the treatment of ulcer of the stomach and duodenum. The alkaline formulæ given in most of the monographs upon diseases of the stomach contain them as part of the prescription. In my opinion they should always be given separately, for the simple reason that whilst the

dose of alkali must be varied from time to time according to the amount of acid in the stomach requiring neutralisation, that of the belladonna must be estimated by the reaction of the patient to it.

As regards the peptic digestion, it is quite possible to limit this artificially to a certain extent. Sailer and Farr* have recently shown us that normally the peptic digestion is inhibited after a certain time by products of digestion, the most important of these being albumose, maltose, and gelatin. Indeed, Danilewsky asserts the presence of a definite body which he calls anti-pepsin, but this has not yet been successfully isolated. If obtainable it should certainly be useful in the treatment of gastric and duodenal ulcers. In the meantime it will be sound practice to include in the diet albumose, maltose, and gelatin. Albumose forms the bulk of the commercial advertised so-called peptones; maltose is to be found as the malt extract of the shops, and may usefully replace other sweetening agents; and gelatine can be easily obtained. In my own practice I invariably order a strong stock which sets into a firm jelly on cooling, to be made from knuckle of veal and cow heel, and employ this in preference to the gelatine of commerce of unknown origin. Veal should be used instead of shin of beef in making our jelly, as our object is to produce an article as flavourless as possible, and as free as may be from the extractives which are such powerful exciters of the gastric secretion.

We may diminish the actual secretion of gastric juice by excluding the physiological gastric juice exciters from the diet, by giving food-stuffs which inhibit gastric secretion, and by the use of drugs.

The physiological exciters of the gastric juice are: (*a*) the psychical agents, viz. the flavour of the food and the act of mastication, and (*b*) those which act by direct contact with the gastric mucous membrane.

The act of mastication is one of the most powerful emotional exciters of the gastric secretion, especially if the food be sapid and tasty. The deduction is that in the dieting of cases of ulcer of the stomach and duodenum we should make use of meats which contain the least amount of flavouring extractives, such as the more gelatinous parts of veal, calves' head, tripe, and chicken, and cook them in such a manner (boiling or steaming) as will not develop the flavouring principles which they do contain. Secondly, that the food should be presented to the patient in such a finely divided condition as to require little or no mastication. We can do this in practice by the preparation of purees and panadas. The simplest panada is ordinary bread sauce, which may be given to a patient with duodenal ulcer after the second or third day, often from the very first. Properly made it is a delicate and appetising dish, and the pheasant or partridge which would accompany it under happier auspices may be conjured up by the imagination of the patient. Panada of chicken, our mainstay in the treatment of many gastric affections, is made by taking a raw fowl, boiling or steaming it until tender, cutting the meat from the bones, passing three times through a mincing machine, pounding in a mortar to a paste with some bread soaked in milk, sieving and warming with a sufficient amount of veal stock to bring it to the consistency of the aforesaid bread sauce.

Unfortunately patients usually get very tired of panadas after a few days, and ask sorrowfully whether it would not be the same if they chewed the food thoroughly. It should be explained to them that this is exactly what we want to avoid, and that the very tastelessness and insipidity of the food is an important part of the treatment. It is needless to say that Fletcherism is the worst possible method of eating for patients with gastric or duodenal ulcer.

Of the exciting agents which act locally upon the stomach-walls, alcohol is the one to which our attention should be especially directed. I feel sure that it should be at once and permanently eliminated from the diet schemes of patients who have now, or have had, undoubted ulcer of the stomach or duodenum. In 1906 L. Kast had an opportunity of making a series of experiments similar to the classical ones of Pawlow on dogs upon a patient with a gastric fistula, which may be said to have finally settled the subject. He found that the stimulating effect of alcohol upon the stomach was greater than that of water or any known food-substance, and that not only the amount, but also the duration of the secretion was affected. The repeated use of even the weaker dilutions of alcohol leads to hypersecretion, which

* 'Amer. Journ. Med. Sci.,' January, 1907.

is followed, sooner or later, by secondary catarrhal processes, these in their turn producing diminished secretion of hydrochloric acid. The habitual consumption of stronger alcoholic solutions leads to an incurable chronic catarrh.

Of the food-stuffs which inhibit the gastric secretion the most important is fat, which markedly diminishes the HCl content. The amount of fat which can be included in the diet depends upon the individual, some patients not being able to stand more than a very small quantity. In the cases under consideration all roasted or baked meat fat must be avoided on account of the irritating principles which are developed by these methods of cooking. As a matter of fact, we should confine ourselves to fresh butter, olive oil, and pancreatised emulsion of mutton-fat. Olive oil may be given just as it is out of a glass, and many patients get to like it after a while. A very good way is to freeze it hard and give in the form of small balls the size of bullets. It is needless to say that the very best genuine olive oil should be used. Many of the failures met with in practice are due to the use of the cotton and other inferior oils masquerading in commerce as "best Lucca salad oil." Cod-liver oil may sometimes be used when other oils are refused, especially when given in the form of an emulsion, which must be made extemporaneously by the pharmacist. The cod-liver oil emulsions of commerce should be tabooed, as not only do we not know what excipient has been used in their manufacture, but they invariably contain hypophosphites, which irritate the stomach.

Cod-liver oil and malt extract may sometimes be usefully given in conjunction, combining as they do two inhibitors of gastric activity. In my own personal practice I invariably make use of Savory and Moore's pancreatic emulsion of fat if the patient can be induced to take it, and have been usually satisfied with the effect produced.

In devising a diet for the treatment of ulceration of the duodenum, we must not shut our eyes to the fact that we are not always able to follow the theoretical considerations without modification. Occasionally we meet with people who are not able to take even an ordinary amount of fat, and after trying it in all available forms are obliged to abandon the attempt.

A strong argument in favour of giving fat in a pancreatised form, in addition to the considerations which I have mentioned, is afforded by what we know of the mechanism of pancreatic secretion. In the ordinary way, as soon as a small amount of the acid gastric contents passes into the duodenum the pylorus reflexly closes. The acid liquid now excites the secretion of the pancreatic juice, bile, and succus entericus. As soon as its acidity has been neutralised by the alkaline succus entericus, the pylorus again opens and allows more acid liquid to enter the duodenum.

It thus follows that if we artificially reduce the acidity of the gastric juice we must diminish its power of exciting the pancreatic secretion, and may consequently expect this to be deficient. We have then a strong indication to supplement its work by the provision of ready pancreatised fats, supplying in a readily assimilable form the carbohydrates which are necessary for the nourishment of the body and the preservation of its weight.

Finally, we can reduce the secretion of gastric juice by means of narcotic drugs. Both morphia and belladonna have this effect, and have been advocated in the treatment of duodenal and gastric ulcer. Morphia, as was pointed out by Hitzig, in large doses, as taken by morphinomaniacs, entirely abolishes temporarily the HCl secretion. Riegel, however, has demonstrated that small doses, such as we are able to give to a patient without narcotising him, have precisely the opposite effect, and act as a gastric stimulant. Any form of opium, therefore, is a mistake in the treatment of duodenal ulcer, and should never be necessary even to relieve pain, as in chloretone, menthol, and anæsthesin we have agents which will serve our purpose.

Belladonna diminishes the acid contents of the gastric juice in all doses, is apparently free from any serious drawback, and has another property which renders it of the greatest service in the treatment of ulcers of the stomach and duodenum. Not only does it diminish the hydrochloric acid, but it has the power of inhibiting the contraction of unstriped muscular fibre, and, in consequence, of relieving spasm of the pylorus. These two properties render it almost indispensable in the treatment of the class of affections under consideration.

Belladonna is best given in the form of pill, and may in some cases be combined with menthol or extract of valerian. Atropine, which is to be preferred in many cases, should be given hypodermically in as large a dose as the patient can tolerate night and morning.

D. *Secure the Stomach against Distension.*

One of the chief ways in which a gastro-enterostomy conduces to the healing of a duodenal ulcer is by preventing distension of the stomach. We can secure much the same result—

(*a*) By preventing pyloric spasm by the administration of atropine and alkalies.

(*b*) By giving the food in small amounts at comparatively short intervals.

(*c*) By giving the drink, which should be hot water, not with the meals, but when the stomach is empty. It will then pass quickly out of the stomach, and will incidentally wash and cleanse the surface of the ulcer in the duodenum.

F. *Treat any Adhesions.*

The recognition of adhesions as a complication of ulcer of the duodenum to be diagnosed and treated has not yet received the attention which it deserves. The probability of any given case being complicated by adhesions is very great if the patient complains of a dragging pain, either when the stomach is quite empty or when it is distended, which is aggravated by movements of the trunk, such as stretching the arms above the head. I am quite sure that a certain number of so-called hunger-pains are merely due to the dragging of an empty and contracted stomach upon an adhesion between the duodenal ulcer and a neighbouring part. Until the discovery of thiosinamine surgical interference was the only method of treatment which held out any prospect of relief. Hebra in 1892 demonstrated that this drug was capable of softening cicatrical tissue, but it was prevented from coming into general use from the fact that it was insoluble in water, and that the alcoholic solution gave rise to pain when injected of a more or less severe character. We have now at our disposal several soluble compounds of thiosinamine which are painless when injected, among which we may mention fibrolysin with salicylate of soda, and tiodine in which it is combined with iodine. I have latterly used tiodine in several cases with very favourable results. The drug is best given by injection into the buttock for five or six days in succession, and afterwards upon alternate days, until at least a dozen doses have been used. If after this period of time no good results have ensued its use should be abandoned. If, however, any perceptible improvement has taken place, it should be continued for some weeks longer.

These, then, are the principles upon which a patient with duodenal ulcer should be treated, and I feel sure, and my experience confirms my belief, that if intelligently applied we shall succeed in the majority of cases in healing the lesion. One of the main points, and incidentally the chief difficulty, is to get the patient to continue the treatment long enough. I am certain that two weeks in bed are absolutely essential, and three would be better. We must bear in mind that the fact of pain having disappeared does not show that the ulcer has healed, for as soon as the ulcer gets into a healthy state and capable of healing, as a rule all pain will cease as in the case of most other ulcers. The actual healing and regeneration of the surface of the open wound must of necessity take much longer. After the period in bed the patient may be allowed to get about, but should lie down for an hour after each meal and should not resume his business for at least a couple of weeks longer, by which time his diet will have been gradually increased to something like normal. Whilst in bed the patient should be measured for a belt to support the lower abdomen and take off any drag from the duodenum. This should be ready by the time that he resumes his ordinary method of life. In order to prevent relapses it is necessary for the patient to observe the following rules for a considerable time after his cure:

(1) A proper belt to support the lower abdomen should be constantly worn.

(2) A daily dose of olive oil should be taken fasting every morning provided that it agrees.

(3) A daily dose of normal horse-serum should be taken after the principal meal for a month, and then twice a week for another month. In the spring and autumn a dose of serum should be taken twice a week for a month as a prophylactic.

(4) All vegetables should be put through a sieve.

(5) Preference should be given to white meats, and to boiling or steaming instead of roasting or baking. All made dishes, curry, condiments, pickles and vinegar should be permanently excluded from the diet.

(6) The blood should be examined periodically for hæmoglobin and steps taken to keep it up to a normal standard.

(7) If there has been any melæna or occult blood the patient should have his stools examined periodically after a three days' hæmogloblin-free diet.

I see no valid reasons why the probability of a permanent cure should not be as great as after a gastro-enterostomy. It is quite erroneous to suppose that there is something magical in an operation which can remove the constitutional condition which allows the formation of the ulcer. The operation, by providing an additional outlet from the stomach, prevents the possibility of distention arising whenever the pylorus becomes spasmodically contracted, and by allowing a regurgitation of bile and pancreatic juice into the stomach diminishes the acidity of the gastric juice. It is difficult to see what more it can do. And as we can imitate these effects almost entirely by the methods which I have outlined, there is no reason why the result should not be the same. Furthermore, we know that anastomotic openings do not keep open indefinitely, and there is no reason which I can perceive why a patient whose ulcer has been cured by operation should not be after a few months in exactly the same position with respect to recurrence as one in whom the healing has been brought about by non-surgical means. And in the former case there is of course the added risk of a jejunal ulcer during the whole time that the anastomosis remains open.

August 8th, 1910.

A CLINICAL LECTURE

ON

RETENTION OF URINE.

Delivered at the London Hospital.

By HUGH LETT, M.B., F.R.C.S.,
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THE causes of retention of urine may be classified as follows:

I. *Obstruction.*
- (1) Within the urethra.
  - Phimosis.
  - Calculus.
  - Foreign bodies.
  - Papilloma of bladder.
- (2) In the wall.
  - Atresia and stricture (congenital).
  - Acute inflammation of the urethra, *e.g.* gonorrhœal.
  - Rupture of the urethra.
  - Stricture (acquired).
  - Tumours of the urethra, *e.g.* epithelioma.
  - Carcinoma of the bladder.
  - Acute atony from over-distension.
- (3) From without.
  - String tied round the penis.
  - Perinæal abscess.
  - Peri-urethral hæmatoma.
  - Tumours of the prostate—simple, malignant.
  - Abscess of the prostate.
  - Pelvic tumours *e.g.* retroverted gravid uterus, uterine fibroids, ovarian tumours.
  - Carcinoma of cervix spreading downwards and involving the urethra.

II. *Nervous causes.*
- (1) Disease of the central nervous system. *e.g.* Tabes.
  - Acute myelitis.
  - Gumma.
  - Tumours.
- (2) Fractured spine.
- (3) Surgical operations in the neighbourhood of the bladder, especially on the rectum.
- (4) Hysteria.
- (5) Spasmodic stricture.

Many of these conditions are rare, and I need not detain you by considering them further, except in one or two instances. Papilloma of the bladder is uncommon as a cause of retention, but I have had one case under my care. He was a man, æt. 45 years, and complained of nocturnal incontinence. The bladder was considerably distended; on percussion dulness extended up to the umbilicus. I found a huge villous papilloma in the bladder growing from the neighbourhood of the left ureteric orifice; it extended over and projected into the internal meatus of the urethra and so prevented a free outflow of urine, and occasionally caused a sudden stoppage of the stream. The growth was removed by supra-pubic cystotomy, and the patient made an uninterrupted recovery with complete control over the bladder.

Calculus is one of the most frequent causes of retention in very young boys; when it occurs in them it is usually formed in the kidney, passes down the ureter and on through the bladder, but is too large to escape from the urethra. It is usually stopped in the prostatic or bulbous portion or at its anterior extremity just behind the meatus.

Occasionally a calculus in the urethra causes retention in adults. I have seen three cases illustrating the different ways in which this may occur. The first was a gentleman who had been operated upon for stricture some years previously by external urethrotomy. An attack of retention of urine occurred. The stricture had contracted down tightly, and immediately behind it was a good-sized phosphatic calculus which had apparently been formed in the floor of the urethra in the site of the operation wound just behind the stricture, though of course it may have descended from the bladder and been stopped by the stricture.

A patient, æt. 62 years, affords the second illustration. He suffers from prostatic calculus, and every now and then finds that he is unable to pass water. On two occasions he has found that a short journey to his medical man in a four-wheeled cab has altered the position of the stone and so relieved the obstruction. On other occasions the passing of a steel dilator or simply a catheter has met the difficulty and pushed the calculus back into the pocket whence it came.

The third patient has a moderately enlarged prostate. I was asked to see him one day during an attack of retention. A No. 7 *coudeé* catheter

was passed as far as the prostatic urethra; there was a slight hitch, and then the catheter went on easily and relieved him. Some hours later he passed water naturally, and in it was a small uric acid calculus, which presumably had caused the obstruction.

Mr. Buckston Browne has kindly given me permission to add the following remarkable case. A gentleman, æt. 69 years, consulted him in September, 1890. He had been treated for some time as a case of spasmodic stricture, and was passing practically all his urine by catheter. Sometimes he could pass it naturally, then suddenly the stream would stop and he would be unable to pass any more. He preferred to use a large catheter, and found that as soon as he had introduced it for four or five inches the urine would flow freely through it. A soft olivary bougie No. 17 French was passed easily, but when withdrawn it was seen to be scratched. This, with the symptoms, suggested that the difficulty was due, not to a spasmodic stricture, but to a calculus in the urethra. The diagnosis was confirmed by examination with the sound.

The calculus was extracted by a scoop, and the patient made an excellent uninterrupted recovery.

Rupture of the urethra is generally the result of a blow on the perinæum, such as a kick, or falling astride a bar or beam. Sometimes it is met with as a complication of a fractured pelvis. The condition found will depend largely on the nature of the injury. When associated with a fractured pelvis the urethra is not infrequently torn across and displaced. On the other hand, when it is due to a blow a hæmatoma rapidly forms in the perinæum from rupture of some of the numerous peri-urethral blood-vessels, and the mucous membrane of the urethra is lacerated; in severe cases it may be pulped. The difficulty offered to the introduction of a catheter increases with the severity of the injury. In slight cases the catheter may be passed readily, but in more severe cases the peri-urethral swelling alters the line of the urethra, so that the catheter is likely to leave the urethral canal and to pass through one of the lacerations into the surrounding tissues. If the urethra is pulped, or if it is torn across, catheterisation is generally impossible.

Acute atony from over-distension is fortunately rare in young subjects, though bad cases are met with from time to time in which complete recovery is delayed, though the patient generally gets well ultimately. Patients who have a simple enlargement of the prostate are much more liable to suffer from this. Many such date their entry into catheter life from a long railway journey, during which they had no opportunity of emptying the bladder. The bladder became more and more distended, their discomfort increased, and finally, when an opportunity did arrive, micturition was not possible.

Spasmodic stricture is usually due to an involuntary contraction of the constrictor urethræ muscle, but it may occur exceptionally in the penile urethra. In its complete form the patient is unable to empty the bladder and has complete retention; when less marked there is considerable delay in micturition and the muscle contracts spasmodically from time to time during the act, so that the stream is intermittent. The patients are highly organised, nervous men, and the condition is greatly exaggerated under certain circumstances, as, for instance, between the acts of a play when the demand for urinals is greatly in excess of the supply, and the subject finds himself at the head of a queue of more or less impatient individuals waiting to take his place. Some find themselves unable to empty the bladder voluntarily while they are on board ship, others while they are on a train. It is important to remember that there is no organic lesion in the urethra; the urine is clear and a full-sized steel dilator can be passed. There may be a little resistance at first, but this soon yields, perhaps with one or two little jerks if steady, gentle pressure is applied, and on withdrawing the instrument it slips out easily and is not gripped as in the case of an organic stricture. If the patient is given an anæsthetic the stricture disappears.

The retention which follows operations upon the rectum is reflex and of a similar nature.

Diagnosis.—Before attempting to treat a case of retention of urine, the cause should be determined if possible, and not only this, but it should be quite clear that the patient is suffering from retention. The two following cases will perhaps illustrate my meaning: A middle-aged man walked into a doctor's surgery and said he thought he had something the matter as he was passing hardly any urine, and he fancied he had got a stricture. The doctor tried to pass a small instrument but failed. He then sent him to hospital with a note as a case of stricture. The house-surgeon saw the patient, read the

note, and tried to pass another small instrument, but only succeeded in entering a recent false passage. The patient was admitted and sent on to the surgeon, who was in the house at the time. He first percussed the abdomen and found no dulness above the pubes, showing that there could not be a very large amount of urine in the bladder, and then took a No. 10 English steel dilator, introduced it into the urethra, and found that it slipped into the bladder by its own weight! There was no retention, and if a medium instrument had been passed in the first place it would not have been caught in a little pocket or fold in the urethra, as the small one probably was, the diagnosis of stricture would not have been made, and the patient would have been spared a false passage and much discomfort. Secondly, the absence of dulness above the pubes should have been ascertained.

In the second case, a man of about 60 was admitted to hospital complaining of pain in the lower abdomen. He said he had not been able to pass water. Abdominal examination revealed a central rounded tumour in the hypogastrium, extending half-way up to the umbilicus; it was tender on palpation. The patient was obviously ill, but the temperature was only 99° F., and the pulse 100. A catheter was passed but no urine was drawn off, or at any rate only a few drops. A diagnosis was accordingly made of retention, probably due to enlargement of the prostate. Further examination, however, by a man of wider clinical experience, proved the case one of a central appendicular abscess, with suppression of urine! In this case if a rectal examination had been made it would have certainly cleared up the diagnosis, quite apart from the assistance which would have been obtained from a more careful investigation of the history, and from a proper appreciation of the result of the examination of the bladder with the catheter.

On the other hand a distended bladder may be overlooked and the abdominal swelling mistaken for an ovarian cyst or a pregnant uterus. This is the more likely to be the case, as the patient will frequently say in answer to inquiry that a large quantity of urine is being passed. If pressed for further details we shall probably learn that it is not really a question of quantity but of frequency, that there are frequent calls, but only a very small amount is passed at each act, or, in still worse cases, that it is constantly dribbling away.

It is important to remember that in women retention of urine is not an uncommon sequela of operations upon the abdomen. It is frequently insidious in onset, as the patient is able to pass a certain amount and so misleads an inexperienced nurse. Before long, however, the patient complains of pain in the lower abdomen, and the condition is recognised, though sometimes not before the bladder has become so distended as to form a considerable abdominal tumour.

The commonest causes of retention of urine in women are bruising of the peri-urethral tissues during labour, and incarceration of certain swellings in the pelvis, such as a retroverted gravid uterus, and ovarian and fibroid tumours.

In men the most frequent causes are stricture of the urethra and prostatic enlargement, and as treatment is somewhat different in the two cases a definite diagnosis should always be made. This can be done easily and rapidly by gently passing a moderately large instrument into the urethra and ascertaining the distance of the obstruction from the meatus. If this is more than six inches there is no stricture in front of the triangular ligament, and the obstruction therefore lies in the prostate with but few exceptions. Enlargement of the prostate is seldom met with in patients under fifty, but on the other hand, stricture is not infrequently seen after this age.

Rectal examination alone is not enough, for though it will reveal enlargement of the prostate towards the rectum a projecting middle lobe cannot be detected, and bimanual examination is, of course, impossible. But even if the prostate is found enlarged, it is not necessarily responsible for the retention, for a stricture may be present as well.

Lastly, disease of the central nervous system must be borne in mind as one of the causes of retention. Prostatectomy has been performed under the impression that the patient was suffering from prostatic retention, and the fact that it was tabetic in origin entirely overlooked. Needless to say the patient's condition was not improved.

Treatment.—A calculus in the urethra must be removed, but the urgent symptoms of retention can often be relieved by passing a small gum-elastic or olivary catheter by the side of the calculus. Sometimes, as in Mr. Buckston Browne's case, the stone can be pushed on one side, and a large catheter passed. If, however, the urine cannot be

drawn off in this way the calculus must be removed. If it has not passed the membranous urethra an attempt should be made to push it back into the bladder, whence it can be removed by the evacuator. If it is in the anterior urethra an attempt should be made to remove it with a scoop, or forceps. If this is unsuccessful, it will be necessary to perform an external urethrotomy and extract the stone through the opening thus made.

Ruptured urethra is a condition of extreme importance, and treatment is directed towards three objects: first, to relieve retention; secondly, to treat the extravasation of urine if it exists, and to prevent its occurrence if it does not; and thirdly, to promote rapid healing with a minimum formation of scar tissue, thus possibly preventing the subsequent development of a stricture, or at any rate diminishing the prospects of a dense intractable one. If extravasation has taken place no one will question the urgent need for free incisions and drainage, but otherwise opinions still vary as to the relative merits of tying a catheter into the bladder if one can be passed, and perinæal section with or without suture of the urethra. The practice of tying a catheter into the bladder, when this is possible, is gradually falling into disfavour, first, because of the danger of death from sepsis in the absence of drainage, and secondly, because some of the worst cases of stricture have followed this treatment. The ideal treatment is suture of the urethra, drainage of the bladder by a tied-in catheter, and I have had some excellent results in this way. It must be remembered, however, that the sutures may give way, and then the patient will not be much better off than if a simple incision had been made in the perinæum. An alternative method of treatment strongly advocated by the late Mr. Reginald Harrison is to make a perinæal incision, expose the lacerated urethra and drain the bladder by a catheter passed through the proximal end of the urethra into the bladder, the other end of the catheter being brought out through the perinæal wound. I have seen an excellent result follow a perinæal incision and supra-pubic drainage of the bladder. No case of contusion of the urethra should be treated lightly. I remember one patient who had a blow on the perinæum which was followed by occasional retention, so that a catheter had to be passed six times in five days. On the sixth day, as he had had no difficulty for twenty-four hours he travelled up to London by train. On arriving in London the retention recurred, extravasation took place, and all attempts at catheterisation failed. In this case the train journey was doubtless responsible for disturbing the blood-clots, and so causing fresh hæmorrhage.

When a patient with an organic stricture of the urethra has an attack of retention, there are usually three factors at work: First, there is the actual narrowing of the urethra due to stricture itself; secondly, there is congestion; and thirdly, spasm, and it is the supervention of the second and third of these which finally determines the attack.

Before attempting to pass a catheter, the position of the stricture should be determined by introducing a full-sized instrument into the urethra and noting how far down the obstruction lies. A small olivary-headed catheter is then taken and an attempt made to pass it through the stricture and on into the bladder, and if this is not successful smaller ones should be taken until the stricture is successfully negotiated. Sometimes a very small gum-elastic catheter goes more easily. If all attempts prove unsuccessful the patient may be given a full dose of tinct. opii and placed in a hot bath. This treatment is often successful in diminishing the spasm and congestion to such an extent that the patient is able to pass a sufficient quantity to relieve him, though the bladder is not completely emptied. But the acute condition is over, and the patient empties the bladder more and more completely at each subsequent attempt. If, however, the hot bath and opium have not the desired effect, another attempt should be made to pass a catheter. If still unsuccessful it will be necessary either to aspirate the bladder by a supra-pubic puncture, or to give an anæsthetic. When the patient is under the anæsthetic it may be possible to catheterise the bladder, but failing this an internal or external urethrotomy must be performed.

One golden rule must ever be borne in mind in attempting to relieve retention by catheter: always practise extreme gentleness; undue haste and roughness are likely to defeat their own ends by making false passages. This not only means that there will be more difficulty than ever in making any instrument enter the stricture, for the point will have a great tendency to engage in the false passage, but the sight of blood coming from the meatus and the

further delay will increase the mental distress of a man who is already in great pain.

When catheterisation has been difficult it is advisable to tie in the catheter. This will not only do away with the possible need of further catheterisation some hours later, but it will also dilate the stricture, and pave the way for further dilatation, either continuous or intermittent. It has been said that there is no such thing as an impassable stricture, and that if urine can get out some form of instrument ought to get in. The truth of this statement is in direct proportion to the experience and skill of the operator.

Retention due to prostatic enlargement is an important condition and must never be treated lightly. Instrumentation of the male urethra for the first time is never a trivial matter, and in the case of old men, particularly those who are known to have chronic interstitial nephritis, and those in whom the efficiency of the kidneys is an unknown quantity, it must be considered an operation of the first importance with a definite, though happily under favourable circumstances, very small, mortality. Every precaution should be taken against exposure to cold. The catheter should be passed in the patient's bedroom if possible. He should be put to bed and kept there for several days until he appears to be perfectly well and all danger is passed. He may show no signs at all, but there will very probably be some symptoms of renal inadequacy, such as a dry furred tongue, thirst, very possibly a slightly quickened pulse, a rise of a degree or so in his temperature, constipation, and loss of appetite. These signs will probably appear for the first time on the day after the catheter has been passed, and may persist for four or five days. The diet should be light, the bowels opened by a dose of calomel or blue pill, and a prophylactic course of urotropin or other urinary antiseptics given.

Another matter of considerable importance is the amount of urine which may safely be drawn off at the first sitting. There is perhaps a temptation to empty the bladder completely, and undoubtedly the patient himself will take a certain pride in knowing that his bladder has contained such a large amount as may be present; his friends, moreover, will yield him a certain amount of sympathetic admiration. But to do so adds unnecessarily to the risk. In a certain proportion of cases the first attack of acute retention supervenes upon chronic retention. That is to say, the bladder may not have been completely emptied for months or even years, and a large quantity of residual urine may have been constantly present unknown to the patient. The danger of completely emptying the bladder at one sitting is twofold. In the first place the sudden withdrawal of the fluid causes a corresponding diminution in the intra-abdominal tension and consequent dilatation of the splanchnic veins. This may lead to, and has actually caused, syncope and death. This may be obviated to a certain extent by passing the catheter while the patient is lying down. In the second place, if there is a large amount of residual urine the kidneys must have been subjected to considerable back-pressure over a long period, and probably show signs of it in dilatation of the pelvis and those changes which are grouped together under the term "chronic interstitial nephritis." The ureters are also dilated, and rapid evacuation of the bladder means sudden relief of the back-pressure on the kidneys with serious results. A satisfactory way of dealing with these cases is to draw off a pint every four hours until the bladder is completely emptied.

The most convenient form of catheter is the coudée; the straight olivary catheter is likely to run into the posterior wall of the prostate, and is more likely to be unsuccessful than any other. The soft rubber catheter is often effective, but it, too, may hitch against the posterior wall, or the middle lobe projection if present, and further, if the wall of the prostatic urethra is much encroached upon by the growth this form of catheter is too limp to be pushed past the obstruction.

A silver catheter can often be passed successfully when other instruments have failed, but it is not advisable to use it as a routine, as it is more likely to cause bleeding and hurt the patient than a coudée or rubber catheter. It has the advantage, however, of being readily sterilised by boiling. Catheterisation ought to be successful in all cases of prostatic retention, and a No. 7 English coudée catheter will generally enter the bladder without difficulty. A bi-coudée will sometimes prove useful in difficult cases. If, as occasionally happens, prostatic retention is complicated by the presence of a stricture in the anterior urethra, a curved olivary catheter will be of great assistance.*

Aspiration by suprapubic puncture may be per-

* 'Brit. Med. Journ.,' June 11th, 1910, p. 1418.

formed, and if necessary, repeated; this has been done as many as ten or twelve times during the same attack, but it is not free from risk, as severe cellulitis has followed when the urine has been septic. Although a hot bath may be given to a patient with stricture retention, and has often yielded good results, it should be employed with caution in prostatic retention, for in these cases the patients are old, and there is a danger of syncope.

Asepsis.—I cannot impress you too strongly with the importance of asepsis. Remember that the introduction of a septic catheter into the bladder may mean ill-health for years; and further than this, it has in many cases brought about the death of the patient from cystitis and ascending pyelonephritis when the kidneys are already suffering from the effects of back-pressure. The more severe forms of urinary fever are also the result of sepsis, though there is much ground for considering that the mild forms are the expression of a reflex nervous disturbance.

The catheter must be carefully sterilised. This may be done chemically by formalin vapour, by solution of biniodide or perchloride of mercury, or by steam by means of Pardoe's or Herring's ingenious sterilisers. Silver and soft rubber catheters can of course be boiled. The penis and hands must be carefully cleaned, and finally a sterilised or an antiseptic lubricant used. In catheterising the bladder in a case of a fractured spine, the anterior urethra may with advantage be previously irrigated with a mild antiseptic.

All cases of retention in which the catheter is passed for the first time, or has not been passed for a considerable period, should be given a prophylatic course of urotropine, or other efficient urinary antiseptic. In cases of retention due to acute gonorrhœa every attempt should be made to avoid passing a catheter, for fear of conveying infection to the bladder and setting up a gonorrhœal cystitis. Hot baths and fomentations, combined with morphia and belladonna suppositories, are often effective, but sometimes a catheter must be passed. In this case the patient should be kept in bed on a light and suitable diet; he should be given large doses of the alkaline salts of potash, which could be combined with the liquid extract of santal, and the anterior urethra should be gently irrigated immediately before the introduction of the catheter.

Retention due to hysteria is usually met with in girls, and may necessitate the passage of a catheter, though this is not desirable, and should be avoided if possible. The difficulty may frequently be solved by the administration of a large soap-and-water enema, as hot as can be conveniently borne; when the bowels act the retention is generally relieved.

August 8th, 1910

ON SOME PITFALLS IN THE DIAGNOSIS OF COMMON DISEASES OF THE EYE.

By A. HUGH THOMPSON, M.A., M.D., B.C.

THERE is a good deal in eye-work that it takes a fairly long apprenticeship to become familiar with, *e. g.* the niceties of retinoscopy and the efficient examination of the fundus with the ophthalmoscope. On the other hand, there is a great deal in the diagnosis of eye conditions with which every practitioner ought to be familiar, and which it should be within the power of any qualified man or woman to acquire by a few months' regular attendance at an ophthalmic clinic. For this purpose the *sine quâ non* is the careful observation of what you see with your own eyes and the habit of describing it accurately without jumping to conclusions. I have generally found the novice, if he ventures on an opinion at all, far too ready to jump to conclusions. Let me illustrate this by a few examples.

An elderly man comes and complains that his sight is getting gradually worse both for distant and near objects. The pupils are normal in size and react to light, but show a greyish reflex. The student jumps to the conclusion that he has cataract. If, however, we take the patient into the dark room and reflect light into his pupils from a plane mirror, they prove to be transparent, showing an uninterrupted red reflex, which proves the diagnosis "cataract" to be wrong. The cause of the failing sight must therefore be something else. The fact is that elderly people very often have pupils which look grey to focal illumination without any appreciable loss of transparency in the lenses. If we now test our patient with glasses we are very likely to find that he is hypermetropic. When he was younger he was able to overcome his hypermetropia in distant vision, and possibly even in near, by the exercise of his ciliary muscles. Now that he is old his lenses have hardened, and consequently the amount of work needed to alter their shape is far greater than before. The ciliary muscles are unable to cope with this increased work unaided, but by providing our patient with convex glasses for general use, and proportionately stronger ones

for near vision, we shall enable him to regain full use of his eyes.

While we are on the subject of changes in the cystalline lens, it is as well to utter a warning against the too free use of the word cataract, even in cases where there are definite opacities in the lens, at any rate in the hearing of the patient. There is no disease which the average eye patient dreads so much. It is quite true that the dread is in most cases very much exaggerated, owing perhaps to the fact that the memory of old people goes back to the days when operations for cataract were far more often unsuccessful than they are now, but that is no excuse for subjecting our patients to severe anxiety, which, if not altogether unnecessary, is at any rate very premature. A large number of old people have a certain amount of opacity in their lenses which in the dark room appears as a few opaque striæ in the pupillary area. In such cases we cannot say how long it will be before the opacities increase enough to cause any serious diminution of sight. Indeed, they may never do so, and until they do we ought at any rate to avoid the word cataract unless directly questioned with regard to it; in most cases of this sort it will be enough to tell the patient that his eyes are showing signs of age, and that he may require to consult us again later on.

As a second instance of the danger of jumping to conclusions, let us take a child who has such severe photophobia that it is impossible to get a proper view of his eyes without either a struggle or an anæsthetic. To avoid either of these alternatives we may be tempted to jump to the conclusion that the child has a corneal ulcer, and this condition, due to phlyctenular keratitis, is so common among the children of the poor that in five cases out of six occurring in hospital practice we should be right. In the sixth case, however, we should be wrong. In some of the most obstinate cases of photophobia in young children that I have seen there has been no corneal lesion at all. In these cases terror of the doctor's examination has a good deal to do with it, and for this reason, unless the child's resistance to the opening of his eyes can be overcome at once, it is often worth while to put him under an anæsthetic so that a correct diagnosis may be made. If the cornea is not affected we may have to treat the conjunctiva, but it will not be necessary to use atropine, which, indeed, may do harm. If the cornea is affected atropine in most cases will do good. The two common causes of corneal lesion in children are phlyctenular keratitis and interstitial keratitis. A third possible cause which should always be remembered is pannus, a superficial affection of the cornea generally confined to its upper half, which is in contact with the conjunctival surface of the upper lid when closed, and always dependent on a trachomatous condition of that lid. Trachoma in this country is not common except in immigrant families, and if ordinary precautions with regard to towels, etc., are taken, the risk of contagion cannot be great, as one rarely finds it in children belonging to families in which the older members are free from it. The essential points in the diagnosis of pannus are the superficial position of the vessels, their distribution over the upper half of the cornea, the lower half being generally clear, and the trachomatous condition of the upper lid. Returning to the two commoner affections:—In interstitial keratitis the disease is situated, as its name implies, not on the surface but in the substance of the cornea, which may show only a diffuse haze or may be vascular, the vascularity advancing as a rule from the upper margin in the form of a pink area with a well-defined crescentic margin—the typical "salmon patch." In the great majority of cases, as is well known, the disease is part of congenital syphilis, and in children over seven the notches in the upper central incisors, first described by Hutchinson, will very often be found. After all, however, the most common cause of photophobia, as I have already said, is phlyctenular keratitis, a disease which especially attacks ill-nourished children, and which, while causing practically no cases of complete blindness, leaves a far greater number of individuals permanently incapacitated by impaired sight than any other single disease. But even in cases where the conjectural diagnosis of phlyctenular keratitis may be right, it is necessary to get a complete view of the cornea, for different cases require to be treated on different lines. Comparatively mild cases of ulceration near the corneal margin can often be cured in a short time by yellow ointment and atropine, with cod-liver oil internally. Cases of a phlyctenular ulcer at the end of a leash of vessels being carried every day nearer to the centre of the cornea need prompt

treatment with the cautery, or, better, with pure carbolic acid. For chronic and relapsing cases—and these unfortunately form a large percentage in hospital practice—treatment is of comparatively little avail without a radical improvement in home conditions, food and housing, and here we leave the sphere of the medical man and enter that of the social reformer.

As a third instance where it is not only undesirable, but unpardonable, to jump to a hasty conclusion, we will suppose that a middle-aged patient comes complaining of a painful eye, and the first thing that strikes us is the congestion of his conjunctiva. If we send away such a patient without further examination, with an astringent lotion calculated to cure his conjunctivitis, we run the risk of making one of two very serious mistakes: We may be overlooking a case of iritis, or still worse, we may be overlooking a case of subacute glaucoma. The first thing to do in such a case is to make sure whether the congestion is confined to the superficial vessels of the conjunctiva or whether it exists also in the deep vessels, which are normally invisible, but which, when the ciliary body is inflamed, appear as minute red lines immediately surrounding the corneal margin. The conjunctival vessels are easily emptied by pressure with the finger; the ciliary vessels are not. In any case when the conjunctival congestion is so great that this test cannot easily be carried out, it is a good plan to insert one drop of a solution of adrenalin chloride 1 : 1000, which will empty the conjunctival vessels more effectually than the finger, while leaving the ciliary ones comparatively unaffected. Having determined whether or not there is any ciliary congestion, we ought always to feel the intra-ocular tension. This needs some practice, but it will not do to dismiss the idea of acute or subacute glaucoma from our minds while there is any possible doubt about the matter. In the next place we must observe the size of the two pupils and test their reaction to light, preferably in the dark room. If the pupil of the affected eye is larger than the other one, we think of glaucoma again; if it is irregular or reacts slowly to light we think of iritis, and in this case we insert a drop of homatropine, which in about half an hour will either dilate the pupil fully or else reveal any adhesions that the iris may have formed with the capsule of the lens. If any such exist the diagnosis of iritis is confirmed, and we proceed to attempt to break them down by means of a stronger mydriatic. The best for this purpose is an ointment containing 2 per cent. each of atropine and cocaine. I have often been able to observe recent adhesions broken down under its influence. If, on the other hand, the pupil is fully dilated by the drop of homatropine first inserted, we should always feel the tension again, as in cases of incipient glaucoma the rise of tension is often only perceptible to the finger when the pupil is artificially dilated. Indeed, if there is any suspicion of glaucoma to begin with, it will be wiser to use no stronger mydriatic than cocaine. In any case where the rise of tension takes place we must insert eserine. A drop of $\frac{1}{2}$ per cent. solution is generally sufficient, but a stronger solution may, in some cases, be required, and anyhow, we shall not be happy until we have actually seen the pupil contract under its influence. Glaucoma and iritis are not the only causes of ciliary congestion. Corneal lesions may produce it, but in most cases these will be obvious. A superficial abrasion, however, may easily go undetected unless we insert a drop or two of fluorescine solution (2 per cent.) and wash away the excess with boracic solution. This shows the exact extent to which the corneal epithelium has been injured as nothing else will. Lastly, cases of cyclitis may be due to a systemic infection brought by the blood from some distant focus of infection (*e. g.* teeth or alimentary canal), or due to the poison of gout. In some of these cases little collections of lymph are deposited on the posterior surface of the cornea, where they are best seen with a corneal magnifier, and where they are known in the clinical slang of the out-patient room as "K.P."—short for keratitis punctata—a misleading phrase, since the cornea itself is not affected at all. About the pathology of these cases we have still much to learn, but it is no doubt connected with the secretory functions of the ciliary body. Their treatment is by no means so simple as that of diseases which are confined to the eye itself, but, as we might expect on general principles, in all cases of cyclitis one indication is to rest the ciliary muscle, and the best way of doing this in all cases where glaucoma can be excluded is the insertion of atropine.

August 8th, 1910.

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CONTENTS.

PAGE

*A Clinical Lecture on Arthritis Deformans. By OTTO GRÜNBAUM, M.D., F.R.C.P. 289

*Smallpox and its Early History. By F. M. SANDWITH, M.D., F.R.C.P. ... 294

*A Clinical Lecture. By CLIVE RIVIERE, M.D., F.R.C.P. 302

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A CLINICAL LECTURE

ON

ARTHRITIS DEFORMANS.

By OTTO GRÜNBAUM, M.D.Cantab., F.R.C.P.Lond.,

Assistant Physician, London Hospital.

THE object of this clinical lecture is to teach. Instruction is most likely to be successful when it is dogmatic. This is my excuse for being dogmatic to-day. The lecture need not contain new observations, nor new deductions, but should present concisely the modern views upon disease without hampering the memory of the student by the introduction of many names. This is my reason for omitting to mention many names even when my words almost coincide with those of text-book or paper.

Inflammation of the joints may be due to various causes:

(1) Trauma.

(2) Infection with definite bacteria such as the gonococcus, streptococcus, pneumococcus, and tubercle bacillus.

(3) The toxins of scarlet fever and syphilis.

(4) Gout.

(5) The toxins injected into the body such as exist in horse-serum, and introduced when used as anti-serum.

(6) Disease of the central nervous system such as locomotor ataxy and syringo-myelia.

(7) Alteration in the blood, with hæmorrhage into the joints. This may occur in hæmophilia, in some forms of purpura.

(8) An unknown cause which gives rise to acute articular rheumatism.

(9) An unknown cause which gives rise to a condition which goes by the name of "arthritis deformans."

It is to this last condition that we intend devoting a short time this afternoon.

We have not any very definite record as to the date at which this disease of the synovial membranes and cartilages first appeared, but we know that arthritis deformans is not a disease of mushroom growth such as appendicitis and appendicitic dyspepsia, for some bones of men who lived about 3700 B.C. demonstrate the characteristic changes of this disease.

Arthritis deformans is a chronic disease which usually involves a large number of joints. Some cases of this disease present marked alteration of the bone and cartilage whilst in others the synovial membrane and peri-articular tissues are chiefly affected. For this reason the disease has been subdivided into osteo-arthritis and rheumatoid arthritis. In osteo-arthritis the primary alteration takes place in the bones and cartilages, the synovial membrane being implicated later, whilst in rheumatoid arthritis morbid processes begin in the synovial and peri-articular tissues and the involvement of the cartilage and bony structures occurs at a comparatively late stage of the disease.

We will deal with these two conditions separately, since when well marked they present very different symptoms, so different as to lead some to believe that they are two distinct diseases. We must remember that one may merge into the other, so that occasionally it is impossible to determine under which heading the case should be placed.

Rheumatoid arthritis occurs more commonly in females than males, the proportion being 4 to 1. The disease may begin at almost any age, the majority of cases occurring between twenty-five and thirty-five. Occupation and station in life have no effect. Frequently some debilitating disease such as influenza or septicæmia precedes the onset and apparently lessens the resistance. The disease may have an acute or gradual onset. When acute, pain in the joints starts suddenly and is of considerable intensity. The joints become red, swollen, tender and painful, with considerable effusion, so that at the beginning there is a similarity to acute articular rheumatism. It is true that in rheumatic fever the pain often begins in one joint and passes to another whilst the inflammation finally settles in a third, and the joints in which the symptoms first appeared may escape any gross alteration. In rheumatoid arthritis with acute onset the joints in which the pain first appears are those in which the greatest alteration take place. Some of the joints which are rarely involved in acute rheumatism seem to be specially selected by the poison of rheumatoid arthritis, namely the sterno-clavicular, the temporo-maxillary, and the joints of the vertebræ. In acute rheumatism the temperature frequently reaches 104° F.; in rheumatoid arthritis it may reach 104° F., but rarely rises above 102° F. In acute rheumatism the administration of salicylate of soda reduces the temperature and relieves the pain; in rheumatoid arthritis the pain may be relieved, but the temperature is rarely markedly reduced by this drug. In rheumatoid arthritis the spleen and lymphatic glands are often enlarged; this is never the case in uncomplicated acute articular rheumatism. When the onset is gradual one joint is first affected, often pain without any visible alteration of the joint may be the first symptom, and this may persist for weeks before any anatomical lesion can be found, however carefully the joint be examined. Then other joints become affected, but the interval is as long as a year or two, and in some cases five or six years elapse before the condition becomes general. In rheumatoid arthritis there are a number of constitutional alterations which are not found in osteo-arthritis, and we must deal with these in turn.

Circulatory system.—It is rare for any valvular lesion to occur in the heart in any form of arthritis deformans. This contrasts the disease with rheumatism. In rheumatoid arthritis the heart is frequently rapid, the blood-pressure is normal, and the blood-vessels are unaffected. In osteo-arthritis, however, it is not uncommon to find that there is arterio-sclerosis, but this to a large extent is due to the age of the individual suffering from the disease, and not an effect of the complaint.

Respiratory tract.—With the exception of non-tuberculous pleurisy being somewhat common amongst patients suffering from rheumatoid arthritis, there is no evidence of any special liability to this part of the body becoming involved.

Blood.—Examination of the blood shows that in the majority of cases there is no alteration in the number of white cells in spite of the slight pyrexia; the differential count does not depart from the normal.

Muscular system.—In rheumatoid arthritis the muscles moving the affected joints may atrophy so rapidly as to suggest that there must be some

alteration in the trophic influence. In other cases the atrophy is slow, and may be well accounted for by disuse. Examination of the muscles with the electric current does not show any evidence of degenerative processes, which one associates with altered nerve supply.

Nervous system.—Examination of the nervous system demonstrates only a slight alteration in signs, in that the reflexes are glib; the hands often present a fine tremor. Occasionally there are subjective symptoms, such as tingling and numbness of the limbs, but so far we have not obtained any definite evidence of degenerative processes taking place in this system.

Temperature.—The temperature in chronic cases may be normal, whilst, as was stated above, in acute cases the temperature may rise to 104° F., but usually falls below 101° F. after the first three weeks of the disease.

The skin.—The skin over the joints frequently becomes glossy in appearance, and simulates in many respects that of aged people, and in addition to this pigmentation appears in irregular areas on the face, neck, as well as the hands. This pigmentation may disappear on the articular symptoms clearing up. The palms are usually cold and moist; described by an Irish physician as like those of a fish.

Glandular enlargement occurs in 50 per cent. of cases, the glands that are most often involved being those which drain the joint affected. The spleen is enlarged in 20 per cent. of the cases.

Osteo-arthritis usually begins in people over forty. One joint is first affected, and the constitutional symptoms are extremely slight or completely absent. Let us contrast them with those which we have recorded as common in rheumatoid arthritis.

Circulatory system.—In this complaint, as in rheumatoid arthritis, it is rare for one to find any valvular lesion, but the disease is often associated with degeneration of the arteries, in which condition one finds a raised blood-pressure, hypertrophy of the heart, and accentuated second sound over the aortic area.

Respiratory system.—There is no definite lesion of the respiratory tract associated with osteo-arthritis, but in town-dwellers bronchitis and emphysema is very common after forty, and therefore frequently emphysema and bronchitis are concomitant.

Muscular system.—Very marked wasting, which is so prominent a feature in rheumatoid arthritis, is not noted in osteo-arthritis. It is true that in osteo-arthritis the muscles watse, but wasting of the muscles can be fully accounted for by their disuse.

The nervous system.—The nervous system is, as a rule, not affected.

The skin.—The skin is usually the skin associated with old age.

Having given a description of the disease, we will now consider the alleged causes. Intestinal toxæmia has been suggested by many, and 38 per cent. of cases suffering from this disease complain of dyspeptic symptoms. Herter found that in this disease there was frequently a diminished quantity of hydrochloric acid secreted in the stomach, diminution in the secretion of the intestinal juice, and in some cases an excessive proteid destruction. (The determination of this last must to a large extent be a personal factor, for, as you know, Folin and his school differed greatly from the Voigt school in their determination of the average quantity which is necessary.) Other suggested causes are mental anxiety, exposure to cold and damp, dental caries, nasal infection, infection of mucous surfaces, and pyorrhœa alveolaris. Before discussing the probability of these, it will be necessary to weigh carefully the evidence in favour of the two alleged causes, namely, bacterial invasion, and alteration in the central nervous system. In 1892 Max Schuller stated that he had noted organisms in sections of joints of patients who suffered from this disease. In the following year he described short plump rods showing polar granulation which he had grown from fluid removed from joints in which there had been effusion. When cultures of this micro-organism were introduced into the joints of rabbits, permanent enlargement of the joints and limbs resulted. In 1897 the same author recorded observations in sixteen cases of chronic villous arthritis. In 1906 he states that in 150 cases out of 230 the diplo-bacillus had been isolated. In 1896 Blaxall succeeded in obtaining an organism in eighteen cases of rheumatoid arthritis; this organism was a minute bacillus exhibiting polar staining; when grown in beef broth it gave the appearance of gold dust. It is in the blood in severe cases, not found in the synovial fluid and distended joints due to other

causes. Von Dungern, in a case of periarthritis, found a micro-organism which in many ways simulated the *coli bacillus*, and since then pneumococci, diplococci, and streptococci have been found and described.

Attempts to corroborate some of this bacteriological work have been doomed to failure, and the multiplicity of micro-organisms found tends to make one doubt their specific character. It is true, however, that many different bacteria may produce similar clinical factors, and this is well illustrated in infective endocarditis, in which disease some fifteen micro-organisms have been isolated, and we have reason to believe that these were causal factors of the disease. It is comparatively rare for anaërobic cultures to be carried out, and it is quite possible that some of those joints which appeared to be sterile were really infected by some strict anaërobe.

In favour of the nervous theory of the disease, we might note that frequently the muscles become more atrophied than we should expect from simply disuse, that the skin over the joints becomes thin and glazed, that the reflexes become increased, and that there is frequently an outgrowth of bone similar in many ways to that observed in Charcot's joint. A Charcot's joint we know is due to some lesion of the spinal cord. In a few cases which have been examined in detail alteration of the nerve-cells has been observed, but this must not be taken as conclusive evidence, because after removal of a limb the nerve-cells which had supplied the muscles of that limb are frequently found to be altered in character.

My experience of the disease does not agree with that of several observers. The Cambridge observer finds a local source of infection in some 48 per cent. of cases, whilst a careful observer in London states that he comparatively rarely finds any cause. It must be remembered that the latter's observations are to a large extent on the so-called well-to-do, and he acknowledges that in hospital patients more than 50 per cent. of the cases suffer from pyorrhœa alveolaris. It may be of some interest to you for me to relate the details of a few cases which point towards the infective origin of this disease.

Three or four years ago I used to ask my patients when they had last visited their dentist. I usually heard that they saw him at least twice a year, and that their teeth were in good order. Now I examine the teeth myself, and have often been astonished at the well-marked pyorrhœa in cases which have been asserted to be healthy by their dentists. As long as there is no caries, and the teeth are fairly tight, the average dentist reports that the teeth are normal. So far I have only once met a case of arthritis deformans in which I have failed to find a possible source of infection, not always in the mouth. I might add that this exception had had her teeth removed two years before because of extensive pyorrhœa, and that her joint trouble had since become stationary.

The treatment of arthritis is so manifold that we may safely conclude none is of much avail. Every effort must be made to find the cause. Any focus of infection must be dealt with, and it is wise to make cultures and possibly a vaccine before carrying out any surgical procedure, so that the resistance of the individual which may be low for the isolated micro-organism can be raised by the vaccine before surgical assistance is rendered. Any surgical treatment opens up fresh vessels, and the patient runs the risk of a more severe infection unless the resistance is increased. Arguments have been brought forward that because vaccines have been used in this complaint without leading to cure, therefore they are useless. This argument is fallacious. When the disease has led to abnormal growth of bone, or exostosis, all that a vaccine can do is to lead to an arrest of the disease; one cannot expect the vaccine to dissolve the bone. In early cases improvement is sometimes very marked, and in these the argument may be raised that the patient was not suffering from the disease under discussion. Several years ago I remember listening to an eminent lady doctor, who asserted that the treatment for arthritis deformans was rest in bed, and that she had cured many patients by sending them to bed. She told us that we had to take her word for the diagnosis of early arthritis deformans, for she knew that her patients were about to develop that disease. I felt far from convinced. When, however, there is limited movement, pain, and fine crepitations upon listening to the joint whilst it is being moved, and there is no history of trauma, and the rest of the causes enumerated at the beginning of this lecture can be eliminated, I think it is permissible to diagnose early arthritis deformans.

These early cases, namely, those in which anatomical alterations have not occurred, quite frequently, recover completely on removal of the source of infection and appropriate vaccine treatment.

Massage in my opinion does good in some cases, because it is a method of auto-inoculation. If this were generally recognised massage would not be ordered daily and be carried out in the usual way, but at intervals of ten days to a fortnight. The consensus of opinion is that massage continued for a long time does more harm than good. Hot-air baths alleviate the pain, but do not cure. Hot-air baths followed by massage make the joint more supple temporarily, and combined with treatment by suggestion, are of benefit. The effect of suggestion and massage must not be overlooked. Some years ago I saw a patient suffering from what had been called "chronic rheumatism of the arms." She had been to several foreign watering-places, with only temporary relief. The real cause of the pain in the arms was due to neuritis induced by the cervical ribs. This lady was annoyed at the diagnosis of what she termed "a deformity," and sought advice elsewhere. I heard later that a course of massage cured the patient so effectively that she intended repeating the treatment in a week or two. Here I have no doubt that suggestion played an important part, and you will note that the latter part of the letter was conclusive evidence that it was no real cure, or why should the treatment have to be repeated?

Those who believe that the disease is due to alteration in the nerve-cells of the spinal cord have adopted the treatment of counter-irritation, and they apply blisters along the side of the spine over areas corresponding to the somites in which the nerve-cells are placed, and which are supposed to have a trophic influence upon the joints affected. In some cases improvement has followed which has apparently justified the treatment, but the percentage of cases recovering is so very small that it is not entirely beyond the possibility of coincidence. However, when all else fails, in my opinion it is justifiable to try it.

Change of climate has a remarkable effect upon the disease, and not infrequently six months at the Riviera, or in Egypt, produces an effect that is really astonishing. Personally I have only seen a return to health in cases of rheumatoid arthritis where the peri-articular tissue has been diseased, but I have heard of cases in which it is stated that there has been a considerable out-growth of bone, and that this bone has been absorbed and the joint become as movable as before after a sojourn of two or three months in Portugal.

Treatment by drugs.—If the view held by Herter be correct that the disease is often due to toxins in the blood, and these toxins are manufactured in the intestinal tract, we can understand how intestinal antiseptics may lead to improvement. Guiaicol and its compounds are favourite drugs in this condition, and the frequency with which they are prescribed almost convinces one it must be of some use. The administration of intestinal antiseptics often is combined with lavage of the colon. Iodide of iron, iodides, quinine, and many other substances possess the reputation of doing good, but I have not been able to satisfy myself that any one of these acts specifically upon the disease. The general improvement may be attributed to general treatment, namely, open air and generous diet; not infrequently rheumatoid arthritis is confounded with rheumatism, and the patient is placed upon a limited diet, and it is the alteration from this to a generous diet which in some cases accounts for the increased development of muscle and motility of the joints.

It may interest you for me to detail the method which I personally adopt in investigating a case of arthritis deformans. I first take the history in order to determine whether there were any debilitating causes. I usually have a radiograph taken in order to inform me whether there is any absorption of cartilage or any outgrowth of bone, because if this is very marked one can only hope to arrest the disease and not to restore the joint to health. I then begin the search for a micro-organism which may be causing either direct infection of the joint or the absorption of the toxin. Since pyorrhœa alveolaris is very common in this condition I usually examine the gums, and if these are above suspicion one has to seek for some other source. It is through the mucous membranes that absorption is most frequent, and therefore the sites examined are the nose, throat, urethra and vagina, and if these fail to reveal any local micro-organism one has to investigate the bladder and intestines; bacteriological examination of the urine is sufficient guide as to the condition of the lining of the bladder. The criticism might be raised that many of these mucous surfaces are covered with micro-organisms, and the variety is very great. That is perfectly true, and no importance can be attached to the mere presence of bacteria, and unless some abnormal micro-organism be found together with evidence of diminished resistance of the patient for this bacterium one cannot expect the slightest improvement from vaccine treatment.

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SMALLPOX AND ITS EARLY HISTORY.*

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IT is a curious criticism on the combined civilisation and sentimentality which rule this country that the prejudices of a minority of people are still able to expose the majority to occasional outbreaks of a very repulsive, filthy disease, which we have now known how to prevent for more than 100 years. If we could all make up our minds to be logical and practical, and insist on the regular performance of vaccination and re-vaccination, we should be able to laugh at smallpox and save the money of the ratepayers by abolishing the isolation hospitals which to-day exist for smallpox cases.

In placing before you the *causes* of smallpox, we must at once acknowledge that, by all analogy with other acute infectious diseases, there must be a living microbe which is the cause of the disease. Since 1892 the belief has been gradually growing that the microscopic cause is a sporozoon, something analogous to the parasite of malaria, which was discovered in 1880 by an obscure army surgeon in Algiers, who by reason of this discovery has become famous—Prof. Laveran, now of the Pasteur Institute in Paris.

Unlike malaria, which requires the presence of a mosquito to transmit the disease, smallpox is conveyed chiefly by direct infection from one person to another. So far as we know, the patient is infectious all through the disease, from the very beginning till the last crusts have disappeared from the skin of the soles of the feet.

Actual contact with the patient is not necessary; it is enough to walk into a badly ventilated room inhabited by a smallpox patient. This leads one to suppose that the poison must be inhaled, and it is believed by many experts that it has been conveyed by air from smallpox hospitals to people living within a radius of a quarter of a mile, or even further.

It is possible, however, that we may find that the air itself is not responsible for carrying the contagion, but that it may be conveyed by some such simple means as the common house-fly, which is much more dangerous than people know.

The disease may also be conveyed by clothes or bedding, and even by the clothes or hair of a healthy person who has been in contact with a smallpox patient.

All ages are liable to the disease if not protected by vaccination, and in olden times the chief sufferers were young children, just as is the case with measles to-day.

Smallpox occurs all over the world, but in this country it is chiefly a disease of the winter and spring months, and epidemics show a decided tendency to abate when summer begins.

The latent or incubation stage is often exactly twelve days, but authentic cases have been reported where the disease developed in as short a time as five days or in as long a period as twenty-three days after exposure to the risk.

Brief description.—The onset is sudden, and is characterised by shivering, pain in the forehead and back, nausea, vomiting, giddiness, and general signs of prostration, accompanied by high fever, which abates on the third day when the eruption appears.

Occasionally this primary fever is associated with a temporary rash, but the true smallpox eruption usually appears about forty-eight hours after the first symptoms, and may be seen on the forehead and wrists.

After a few hours the spots become distinctly raised and are felt to be hard, like small shot under the skin; this is the papular stage of the eruption.

The spots prefer some situations of the body to others. Besides the face and wrists, the head, back, hands, feet, inside of the mouth, larynx, and eyelids suffer, while the limbs and some other parts are less affected.

The eruption picks out any portions of the skin which have been subjected to pressure or irritation, so that women may show a large number of spots round the waist or below the knee where corsets and garters have been worn, and if a poultice has been applied to the back for the pain the site will be well marked by the eruption. By the time the papule has become a vesicle the patient feels comparatively well, in spite of his disfigurement and the discomfort produced by spots almost all over him. His fever has gone and his appetite has returned.

After four days of the stage of vesicle the con-

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tents of each spot become pustular, of yellowish colour, and in severe cases many of them run into each other and become confluent.

Inflammation occurs round the pustules, then the secondary fever begins and lasts for about a week. About the ninth day of the eruption or the eleventh of the disease the pustules begin to burst and discharge thick pus, which has a most disgusting smell, though some of them dry up without breaking.

The matter which escapes forms a yellow or brown scab, and in a few days separates from the skin, and if the skin has been much involved permanent pitting or scarring remains.

The milder cases of smallpox in which the pustules do not run together are called "discrete," while the confluent cases are those in which large patches are formed by the mingling of the contents of many spots. Bad cases may be complicated by additional troubles, such as bronchitis, eye diseases, boils and abscesses.

A fatal form of the disease is called "black" or "hæmorrhagic smallpox," in which there are signs of bleeding on the skin and from the nose, gums, and other mucous membranes.

It is possible, but very rare, for an individual to suffer from two attacks of smallpox; in many alleged cases it is found upon investigation that one of the attacks was in reality chickenpox.

Diagnosis.—During the two days of fever before the eruption appears it may be difficult to determine from what disease the patient is suffering, unless everyone is on the special look-out for new cases during a local outbreak. The diseases from which smallpox has to be distinguished at this early stage are chiefly influenza, measles, scarlatina, and lumbago.

After the appearance of the eruption there are still pitfalls for the unwary: chickenpox, acne in its pustular form, glanders, some eruptions caused by drugs, and even cases of severe gnat-bite have been mistaken for smallpox. In 1908 six persons in London certified to have smallpox were found on examination by an expert to be suffering from chickenpox.

The *mortality* of confluent cases, unprotected by vaccination, is 60 per cent., while children under two years of age almost invariably die. Confluent smallpox among the vaccinated is always a serious disease, but those who have been vaccinated and contract the discrete type of smallpox never die unless there is some accompanying complication.

Statistics from all over the world prove that the death-rate from smallpox is from five to sixteen times greater among the unvaccinated than among the vaccinated. Physicians are, perhaps, the best vaccinated class in England, and the deaths from smallpox among them is only thirteen per million, although they come into closer contact with the disease than other members of the population, among whom the deaths are seventy-three per million.

A smallpox patient must be isolated in his own house or kept in a hospital until the last scab separates from the skin. In mild cases this process may be completed in three weeks, but in severe cases it may extend over three months.

Prevention of the spread of the disease.—Directly a case is recognised it should be notified and isolated, usually in a special hospital. The next step is to vaccinate without delay all individuals who have been in contact with the sick person. If, for instance, the disease has appeared in a common lodging-house, the healthy contacts should be kept under observation until their vaccination has taken or until the incubation period since their last exposure is over—say sixteen days. The contacts should have disinfecting baths and their clothes should be disinfected with steam.

In the case of a fixed residential population such quarantine is not so necessary, and it is sufficient for an inspector to call at the house daily to find out if there are any new cases. Disinfection of the rooms occupied by the smallpox patient is of course essential, and for this a formalin spray may be used, after which the wall-paper may be stripped and the walls and stair-cases thoroughly cleansed or whitewashed. The bedding and clothes must be sent to the steam disinfector.

All necessary disinfection should be undertaken by the local sanitary authority free of charge.

If there is any fear that smallpox is going to spread in a district, free vaccination stations should be opened at hours convenient to the public, and leaflets describing the advantages of protection should be distributed.

With regard to smallpox hospitals, the Local Government Board decrees that they shall be erected in isolated places with a population of less than 600 people within half a mile. The nurses in

such hospitals, when granted leave to go outside the grounds, must take on each occasion a disinfecting bath, and their hair must be washed, disinfected, and dried with a drying-box.

The medical men and others who enter the wards must wear overalls and carefully disinfect their hands on leaving. No visitors should be allowed with the exception of ministers of religion and the relatives of hopelessly ill patients. Vaccination should be strictly enforced upon all the staff and visitors.

For Greater London the Metropolitan Asylums Board possesses the following accommodation near Dartford, in Kent: Joyce Green Hospital, 940 beds, with 315 acres of land; Long Reach Hospital, 300 beds; Orchard Hospital, 800 beds; total, 2040 beds. It is satisfactory to know that there are at present only five of these beds occupied, and that all five cases are the direct result of one mild case, which was found walking about until she was removed to the hospital.

The capital outlay expended upon these three hospital buildings, with the land, already represents £573,113, and the necessary annual expenditure for 1907–8, which are the latest accounts published, was £13,464, and in the previous year was £22,151.

Since 1902, when there were 818 cases under treatment, there have been very few smallpox patients. The number of cases admitted to the hospitals of late years has varied from a maximum of 941 in 1895 to a minimum of only one case in 1908.

During the thirty-eight years of the existence of the Metropolitan Asylums' Board 72,091 smallpox patients have been admitted, but statistics show that, whereas during the first fifteen years of the Board's existence London was visited by smallpox every third or fourth year, during the last twenty-three years these outbreaks have only occurred every seventh or eighth year.

Moreover, for the third year in succession not a single death from smallpox has occurred in London.

Early history.—It is impossible adequately to estimate the prevalence of smallpox in days now happily gone by, for though we have accounts of the great epidemics of this dread disease, epidemics when thousands were attacked and thousands died, its everyday occurrence, especially among the poorer classes, was taken so much as a matter of course that the ancient writers barely refer to it. Our memories are very short, and it seems almost incredible to us to think what terrors that word "smallpox" inspired in our forefathers a little over a hundred years ago, for it meant not only the likelihood of death—it meant, if life was spared, probable disfigurement, or possible blindness or deafness, and from this disease, according to medical historians, only one in every seven people born in England escaped. In France and Germany it was the same. A French physician, writing in 1754, says that every tenth death was due to smallpox, and one fourth of mankind was either killed by it or crippled or disfigured for life. A German professor of medicine tells us in 1796 that 65,220 persons died of smallpox in the German-speaking countries in that one year.

The antiquity of smallpox is said to be immense, for it seems to have been known in China and India from time immemorial, and in both these countries the disease was placed under the special protection of a goddess, to whom sacrifices were made and to whom temples were dedicated. Sir Alfred Lyall tells us that the Hindus promote smallpox to the rank of a powerful deity—a goddess with rites, temples, priests, sacrifices, and burnt offerings—and when we understand that the outward and visible marks of smallpox are universally regarded, not only as the signs and symbols of divine displeasure, but as the very embodiment of the deity—the shape which the goddess chooses to assume—when we realise all this we may form some notion of the terror which has been inspired in many generations of Indians by this awful scourge.

In the Ebers papyrus of Egypt, which dates from about 3730 B.C., there is a disease mentioned which is supposed to be smallpox, but the first genuine medical reference to the subject is by the great Arabic physician, Rhazes, who wrote of it more than 1000 years ago as an illness well known all over the East, and described the symptoms and treatment.

In the Koran we read the story of the Abyssinian Christians, who marched upon the holy city of Mecca, with intent to destroy it, when a miraculous flock of birds flew above them and showered small stones upon them from their beaks and claws, utterly destoying the force, and we have little doubt

that these stones that fell upon the invaders were the smallpox pustules. A physician, called Herodotus, who lived in Rome in the time of Trajan, was the first who wrote of smallpox in Europe. Later Galen gave an account of the pestilence, which spread throughout the whole Roman empire between 160 and 168 A.D. Again, we hear of its overrunning nearly the whole of southern Europe in 580, and it is probable that the ancient chroniclers were right when they concluded that the crusaders of some centuries later were often the means of diffusing the infection through every country through which they passed.

It is generally supposed that the original sites of smallpox were in India and in Central Africa, but wherever it originated, there is now no country of any latitude where it has not wrought destruction.

Almost all the writers of the middle ages mention smallpox, but from none of them do we get any statistics as to its extent or virulence; it seems to have been accepted as an inevitable and every-day scourge.

The detractors of vaccination maintain that smallpox is a disease which is dying out of its own accord, at least in Great Britain, that it first left the richer classes, then it left the villages, then it left the provincial towns and confined its attacks to the dwellers in large cities, where overcrowding encourages the spread of infection. But we think that this shrinking of the numbers attacked is not due to any loss of virulence in the disease itself, for where it gets a good footing, among a sufficient number of unvaccinated, and therefore susceptible persons, it has lost none of its deadly and cruel qualities. We best see the destruction wrought by smallpox in modern days when it enters a primitive tribe hitherto free from it, where it finds fresh material and runs riot among the unfortunate people. We have a striking instance of this in North America, where smallpox followed the march of civilisation westward and broke out among the native Indians, first in 1837 and again in 1850, after the arrival of the gold diggers. It reached Vancouver's Island in 1862, and in one winter killed more than a thousand Indians, so that considerable fear was entertained lest the entire native population should be destroyed. In like manner we hear of smallpox reaching Greenland from Denmark in 1733, when almost the whole colony was killed by it. A traveller in the interior of North America, writing in 1837, states that: "The Big-bellied Indians and the Ricarees, lately amounting to 4000 souls, were reduced to less than half. The Assiniboins, 9000 in number . . . are, in the literal sense of the expression, nearly exterminated." The cause of this devastation was an epidemic of smallpox.

It is difficult to determine when smallpox first became widely prevalent in England. The earliest medical writers in this country, John of Gaddesden (14th century) and Gilbert repeat only what Arabic writers had said before them on the subject, but Gaddesden gives us the following prescription for its treatment: "Then let a red cloth be taken, and the smallpox patient be wrapped in it completely, as I did with the son of the most noble King of England (Edward I) when he suffered those diseases; I made everything about his bed red, and it is a good cure, and I cured him in the end without marks of smallpox." But among Arabic writers, and therefore among their European imitators, smallpox and measles were always confounded together, although they made the ingenious distinction of considering the bile to be the originator of measles, whilst the blood had to account for the smallpox. Whether the king's son really suffered from smallpox or whether it was measles we shall never know, though we have no other evidence of smallpox having occurred in England till the sixteenth century.

John of Gaddesden (1280?–1361), who took his name from a little village on the borders of Hertfordshire and Buckinghamshire, was a London priest physician, who attended a son of Edward I in the smallpox, wrapped him in scarlet cloth in a bed; both bed and bed-room were furnished with scarlet curtains.

Dr. Norman Moore has suggested that his contemporary Chaucer may have had John of Gaddesden in his mind when he described his "Doctour of Phisik."

Dr. J. Galloway tells me that the patient was Thomas of Brotherton, Earl of Norfolk (1300–1338), the elder son of Edward's second marriage with Margaret, daughter of Philip III of France.

Peter Martyr, in a letter written on March 3rd, 1514, tells his correspondent that the English King, Henry VIII, had a fever which the doctors feared would turn to the pustules called variolæ, and an

official letter to the English ambassador in the low countries informs him that the English King has lately suffered from the smallpox. One Phaer, wrote, about 1553, a medical work called the 'Book of Children,' in which he writes of smallpox and measles as of one illness, saying, "They be but of one nature and proceed of one cause. The signs of both are so manifest to sight that they need no farther declaration." In all the references to the smallpox in these Tudor days we read that the disease was lightly thought of and apparently needed little treatment.

I will give you one more instance, the narrative of the troubled conscience of Master Richard Allington, a money-lender, who believed himself to be dying in 1561, and summoned lawyers and doctors to his bedside, before whom he made this testimony: "Masters, seeing that I must needs die, which I assure you I never thought would have come to pass by this disease, considering it is but the smallpox, I would therefore most heartily desire you to suffer me to speak until I be dead, that I may discharge my conscience." He continues by mentioning some special acts of usury and offers restitution to the amount of some hundreds of pounds. "And then, he thought he should have died, but then broth being given unto him he revived again and fell to prayer and gave himself up wholly to quietness."

Although we have no official accounts of the smallpox to make us think it was very prevalent, we hear of many isolated cases among well-known people, and it must be remembered that it was the people of quality only whose health received much attention. It was only when their safety was endangered that serious notice was taken of the health of the community, whilst the physicians of the day knew nothing of what was happening among the poorer folk.

Thus Pace writes to Wolsey from Wallingford in 1518, the Court being there, to say that the king, Henry VIII, is leaving "as it is time; for they do die in these parts in every place, not only of the small pokkes and mezils, but also of the great sickness." And again, a letter written in 1591 to a member of Queen Elizabeth's court, says: "Hir Highess wold you should remove from that place where the small pokkes were, to take the fresh and clere ayre the better to purge you from the infection." These letters and others make it evident that smallpox was assuming a more serious aspect and was becoming more prevalent.

In the year 1561 Queen Elizabeth became twenty-eight years old, and had been for three years firmly seated on the throne of England. While she dallied with her favourite, Robert Dudley, her various royal suitors kept from any serious advances. In France there was civil war, hateful persecution in Spain, and in Scotland, where Mary (Queen of Scots) had just returned after the death of her French husband, there was almost anarchy. The Netherlands were filled with deep discontent, ready at any moment to burst into flames. England alone was quiet and prosperous; Elizabeth lived a gay and merry life, always vigilant, alert and prepared for emergencies, such as the demand of Mary Stuart to have her title to the succession acknowledged, or pressure from the Pope to send a Legate to England. Yet in October this country was nearly scared out of its wits, for the Queen became dangerously ill with smallpox, and for some hours at least her life trembled in the balance. When Parliament met the faithful Commons presented a humble petition to her Majesty "to take to herself some honourable husband whom it shall please you to join unto in marriage."

Elizabeth returned an answer similar to her former speech: "This shall be for me sufficient, that a marble stone shall declare that a queen, having reigned such a time, died a virgin." The Archduke Charles still hoped to marry Elizabeth, who favoured, however, Francis duc d'Alençon, a hideous dwarf, blotched, scarred and stunted by smallpox in childhood. His pock-marked face and discoloured skin made him look like a frog when he was seated, and Elizabeth's pet name for him was "Little Frog." She was nearer marrying this deformity than any other suitor, and in the archives at Hatfield there are still preserved more than one hundred love-letters that passed between the two, as amorous as were ever read at a trial for breach of promise.

Two well-known ladies at the court of Queen Elizabeth who were the victims of smallpox were Penelope Devereux and Mary, daughter of the Duke of Northumberland. Penelope was the sister of Queen Elizabeth's favourite, the Earl of Essex, and was a beautiful girl of fourteen when she won the admiration of the learned Sir Philip Sidney, who wrote her love sonnets, calling her Stella and himself Astrophel. Spenser wrote of them:

"To her he vowed the service of his days;
On her he spent the riches of his wit;
For her he made hymns of immortal praise, of only her he sang, he thought, he writ."

Penelope, at the age of nineteen, married Lord Rich, and became the mother of seven children. But not content with that family she afterwards presented three sons and two daughters to Lord Mountjoy, and at the age of thirty-five she was attacked by smallpox, but recovered, "without any blemish to her beautiful face."

Mary, the daughter of the Duke of Northumberland, married Sir Henry Sidney in 1551. She is described as "a full, fair lady, endowed with all womanly and wifely virtues," but she lost her looks by catching the smallpox while nursing Queen Elizabeth through that disease, and thereafter she "chose rather to hide herself from the curious eyes of a delicate time than come upon the stage of the world with any manner of disparagement."

The gradual rise into prominence of the smallpox in the latter years of Elizabeth's reign is remarkable, and with it came the dread of infection, of which we hear less in earlier writings.

In 1604 began the classification of London deaths by the parish clerks, and by 1629 their weekly and annual bills were regularly printed. In the earlier bills "flox, smallpox, and measles" receive one entry, flox signifying confluent smallpox. In 1629 the number of deaths from these diseases was registered as seventy-two, but in 1634 there seems to have been a considerable epidemic, as the number had risen to 1354, and another epidemic took place in 1641, when, we are told, "both Houses (of Parliament) grew very thin by reason of the smallpox and plague that is in the town."

The Royal House of Stuart was peculiarly susceptible to the disease, and furnished instances of dangerous and even fatal attacks. Charles II assumed the Crown in May, 1660, and his two brothers, the Duke of Gloucester and the Duke of York, accompanied him in his journey to England from the Hague. In September of that year the Duke of Gloucester was attacked by illness, and "'tis thought he will have the smallpox." A few days later "the doctors say it is a disease between the smallpox and the measles; he is now past danger of death for this bout, as the doctors say." Later, again, the Duke "is in good condition for one that has had the smallpox," but a few days later his illness took an unfavourable turn. The doctors had left him at six in the evening, apparently satisfied with his condition, but shortly after he bled at the nose, fell asleep, and when he awoke he passed into an unconscious condition, and died on the tenth day of the disease. Pepys, who was always ready to accuse the doctors, attributes his death to their negligence, but as we read that they did not try any of the violent measures sometimes used at that time, but gave him nothing because they considered the case a very favourable one, we need not consider them responsible. Apparently the Stuart family had little stamina to resist severe illness when it attacked them. The next case was the King's sister, Princess Mary of Orange, who came on a visit to the Court a short time after the death of her brother. In December of that year she also contracted smallpox, and died on the 21st. Again Pepys reports that "much fault was laid upon Dr. Frazer and the rest of the doctors for the death of the Princess."

Her sister, Princess Henrietta, who had followed her on a visit to Whitehall, was removed to St. James's on the day of her sister's death "for fear of the smallpox," but she caught it and recovered.

To continue the history of smallpox in the Stuart family, we find it reappearing in the next generation in the family of James II, who lost one son of it besides his daughter, Queen Mary, wife of William of Orange, while the only child of his other daughter, Queen Anne, died at the age of eleven from what some called smallpox and others malignant sore throat.

We have a detailed account of Queen Mary's illness and death in Macaulay's 'History of England,' which you may care to hear.

The King "had but too good reason to be uneasy. His wife had, during two or three days, been poorly, and on the preceding evening grave symptoms had appeared. Sir Thomas Millington, who was physician-in-ordinary to the King, thought that she had the measles; but Radcliffe, who, with coarse manners and little book learning, had raised himself to the first practice in London chiefly by his rare skill in diagnostics, uttered the more alarming words, smallpox. That disease, over which science has since achieved a succession of glorious and beneficent victories, was then the most terrible of all the ministers of death. The

havoc of the plague had been far more rapid; but the plague had visited our shores only once or twice within living memory; and the smallpox was always present, filling the churchyard with corpses, tormenting with constant fear all whom it had not yet stricken, leaving on those whose lives it spared the hideous traces of its power, turning the babe into a changeling at which the mother shuddered, and making the eyes and cheeks of the betrothed maiden objects of horror to the lover. Towards the end of the year 1694 this pestilence was more than usually severe. At length the infection spread to the palace, and reached the young and blooming queen. She received the intimation of her danger with true greatness of soul. She gave orders that every lady of her bedchamber, every maid of honour, nay, every menial servant who had not had the smallpox should instantly leave Kensington House. She locked herself up during a short time in her closet, burned some papers, arranged others, and then calmly awaited her fate.

"During two or three days there were many alterations of hope and fear. The physicians contradicted each other and themselves in a way which sufficiently indicates the state of medical science in that age. The disease was measles, it was scarlet fever, it was spotted fever, it was erysipelas. At one moment some symptoms, which in truth showed that the case was almost hopeless, were hailed as indications of returning health. At length all doubt was over, Radcliffe's opinion proved to be right. It was plain that the Queen was sinking under smallpox of the most malignant type.

"All this time William remained night and day near her bedside. The little couch on which he slept when he was in camp was spread for him in the ante-chamber, but he scarcely laid down on it; the sight of his misery, the Dutch envoy wrote, was enough to melt the hardest heart. Nothing seemed to be left of the man whose serene fortitude had been the wonder of old soldiers on the disastrous day of Landen, and of old sailors through that fearful night among the sheets of ice and banks of sand on the coast of Goree. The very domestics saw the tears running unchecked down that face, of which the stern composure had seldom been disturbed by any triumph or by any defeat. Several of the prelates were in attendance. The King drew Burnet aside, and gave way to an agony of grief. 'There is no hope,' he cried; 'I was the happiest man on earth; and I am the most miserable. She had no fault, none; you knew her well, but you could not know, nobody but myself could know her goodness.' Tenison (Archbishop of Canterbury) undertook to tell her that she was dying. He was afraid that such a communication abruptly made might agitate her violently, and began with much management. But she soon caught his meaning, and with that meek womanly courage which so often puts our bravery to shame submitted herself to the will of God. She called for a small cabinet in which her most important papers were locked up, gave orders that, as soon as she was no more, it should be delivered to the King, and then dismissed worldly cares from her mind. She received the Eucharist, and repeated her part of the office with unimpaired memory and intelligence, though in a feeble voice. She observed that Tenison had been long standing at her bedside, and, with that sweet courtesy which was habitual to her, faltered out her commands that he would sit down, and repeated them till he obeyed. After she had received the Sacrament she sank rapidly, and uttered only a few broken words. Twice she tried to take a last farewell of him whom she had loved so truly and entirely; but she was unable to speak. He had a succession of fits so alarming that his Privy councillors, who were assembled in a neighbouring room, were apprehensive for his reason and his life. A few minutes before the Queen expired William was removed almost insensible from the sick-room."

The affection with which her husband cherished her memory was soon attested by a monument, the most superb that was ever erected to any sovereign. No scheme had been so much her own, none had been so near her heart, as that of converting the palace at Greenwich into a retreat for seamen. While she lived scarcely any step was taken towards the accomplishing of her favourite design. But it should seem that as soon as her husband had lost her he began to reproach himself for having neglected her wishes. No time was lost. A plan was furnished by Wren; and soon an edifice surpassing that asylum which the magnificent Lewis had provided for his soldiers rose on the margin of the Thames. Whoever reads the inscription which runs round the frieze of the hall will observe that

William claims no part of the merit of the design, and that the praise is ascribed to Mary alone.

Smallpox had claimed the brother and sister of Charles I in 1660, and in the following year there was a severe epidemic in London and throughout the country, whilst the fatality of the disease was much remarked upon. In an anonymous essay of that time the blame is laid upon the doctors, as Pepys and his gossips had done. "And I know not," says the author, "by what fate physicians of late have more lost their credit in these diseases than ever; witness the severe judgment of the world in the cases of the Duke of Gloucester and the Princess Royal; so that now they stick not to say that at least in these a physician is more dangerous than the malady."

It is easy to understand how the infection was spread broadcast when we realise that in these days of the seventeenth century there was no isolation of cases, and Pepys tells us in 1668, "It also hardly ever was remembered for such a season for the smallpox as these last two months have been, people being seen all up and down the streets newly come out after the smallpox." If it was so evident to Pepys that these people had lately suffered from the illness, they were doubtless still, in many cases, infectious.

"How is it," asked the great Dr. Sydenham, a generation later, "that so few of the common people die of this disease compared with the numbers that perish by it among the rich?" And Lynn (1714–15) agreed that the mortality was far less heavy among those who could not afford the fees of the physicians and apothecaries than among the idle and pampered. The conclusions of these physicians, practising entirely among the rich, may not have been very accurate; for in those hard-hearted days the poor came and went without their well-to-do neighbours knowing or caring much about them.

It is curious also that the writers of this period speak of smallpox as being a malady which is peculiarly light and mild in children, so that, as one Morton tells us, "neither the helping hands of physicians are called in nor the abounding skill of complacent nurses is put in requisition." On the other hand, however, the keen observer Sydenham speaks of many thousands of infants dying, and it is possible that the very complacency of the nurses accounted for the fact that their deaths were often not returned as smallpox. The cases that attracted attention were those of well-known adults, and this fact may have caused the more casual observer to think that infants escaped.

By the middle of the eighteenth century our statistics had become more accurate, and then we find that the deaths are mostly among children. In a total of 1622 deaths, for instance, only seven were above the age of ten. This peculiarity still continues in smallpox, and this is why it is a matter of such supreme importance to vaccinate infants early in life. In unvaccinated communities it is the children who perish far more than older people, and the statistics prove this in every general epidemic till the year 1837, when the age-incidence rose. This can be accounted for by the fact that a large proportion of our infants had then already been vaccinated, whilst the older people had again become susceptible, not having been re-vaccinated.

The next great epidemic of smallpox after the year in which that disease had robbed England of a good queen was in 1710, in which year 3138 people are reported to have died of it in London alone, and it was followed by another epidemic four years later, which carried off 2810 people. London was then as insanitary as it well could be, terribly overcrowded, and its mortality from all diseases was very high. But constant recruits from the country kept up its numbers, and in these fresh arrivals the smallpox found fresh susceptible material.

Dispensaries were started in various parts of London, in a faint attempt to help the poor, but the evil was too great to be easily remedied.

The richer folk suffered from the epidemics among the poor, for the disease was often introduced into great houses by servants, which gave rise to the opening of the first smallpox hospital in 1746, which was chiefly intended for the servants of the rich, who brought letters of admission. Before this time it had been the custom for servants who contracted smallpox to be sent to nurses who kept houses for these cases. Thus a Lady Wyche, whose butler was attacked, writes that "she knew of an eminent nurse who had managed above twenty of my lord Cheyne's servants in the smallpox, and every one of them had recovered."

I have already referred to the danger of blindness as a complication of smallpox. Surgeon-General Pinkerton gave evidence before the Royal Com-

mission on vaccination to the effect that in 1863 nearly the whole population of Scinde (India) was marked with smallpox, and that blindness resulting from that disease was very common. According to Sir Ranald Martin, 75 per cent. of the blind in some parts of India owed their affliction to smallpox, and in the report on the Indian census of 1872 there is a remark that the large number of blind persons was doubtless principally to be attributed to smallpox. In the parts of India which are well vaccinated blindness has already been reduced one half.

Similar evidence may be obtained from other countries where vaccination is imperfectly carried out. For instance, in the Statistical Report on the health of the Navy for 1864, it is stated that almost every blind beggar in Japan from whom inquiry was made said that his blindness was the result of smallpox.

With regard to this country, Sir J. Blane has stated that during the last half of the eighteenth century fully two thirds of the applicants for relief at the hospital for the indigent blind owed their loss of sight to smallpox.

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August 15th, 1910.

Tuberculin for the treatment and diagnosis of tuberculosis was much in evidence in the Medical Museum at the Annual Meeting of the British Medical Association held in London this year. Any medical man wishing to see tuberculin in full use should visit the Tuberculin Dispensary on a Wednesday or Saturday morning. At this Dispensary the dosage of tuberculin is carried out under Dr. Camac Wilkinson, whose work at Sydney with tuberculin is now well known.

A CLINICAL LECTURE.

Delivered at the Polyclinic.

By CLIVE RIVIERE, M.D., F.R.C.P.,
Physician, East London Hospital for Children.

The first two cases I am showing you to-day are cases of localised tuberculosis treated with tuberculin. Their histories are as follows:

S. C—'s mother died of phthisis. At the age of ten months he had a finger removed for tuberculous bone disease, and soon after exhibited bone disease in both feet and the left arm. He came to me about eighteen months ago after one year's treatment of these conditions, and showed then the following lesions: Tuberculous bone disease of the left foot, with a sinus showing much redness and swelling, an "abscess" on the right foot, a partly healed sinus over the left olecranon process, and a few scars of superficial abscesses. He was put on $\frac{1}{6000}$ mgrm. of human tuberculin T.R., and this was continued with some variation of dosage, with the result that eight months later all these lesions were completely healed, and his weight had risen by 5 lb. The disease has remained healed in all these lesions, but tuberculin has been continued up to the present time, that is, for some ten months since healing appeared to be complete, and he has added a further 5 lb. to his weight. You will see the scars of the lesions, and also a tuberculide which appeared while he was having tuberculin, and on which the remedy appears to have no effect at all.

This is a very ordinary case, and is shown for that very reason; it serves admirably to demonstrate certain points in the treatment of localised tuberculosis with tuberculin. In the *first* place it illustrates the fact that if the disease has been present a long time, rapidity of cure must not be looked for. Here the disease had been present for a year, and the local lesions took eight months to heal up under tuberculin. When the great amount of repair needed to make good such a loss by disease is considered, I think you will admit that eight months is not a long time. I generally warn my patients that the cure will take as long to accomplish as the disease has already lasted, and this is generally a fair estimation.

The *second* point that such a case, but still

better the second case I am showing, exhibits, is the way the general nutrition rises under tuberculin given in suitable doses. In this case the child was fairly well nourished at the beginning, yet the weight rose steadily during the process of cure. If the weight does not rise the dosage is at fault. On the other hand, sometimes a greater effect on the local lesions may be attained by a short course of dosage too high to allow rise of weight, and I do not hesitate to use this means on occasion in a child of sufficient nutrition. It should be done, however, with caution, and never where nutrition is low.

The *third* point suggested by this case is exemplified by the fact that I am still giving tuberculin, though the local lesions have been healed for ten months. Why am I doing this? I am doing it because I presume, I am sure rightly, the presence of an *occult primary lesion*, from which these local areas were infected. Such a lesion is almost certainly a tuberculous gland, and its situation is almost certainly intra-thoracic or intra-abdominal, and quite probably both. Now glandular tuberculosis is very slow to heal; where a glandular abscess has been opened, as in the neck, the dead tissues are removed and plasma containing antibodies flows through the wound and the conditions are favourable for rapid cure, but where a gland in the thorax smoulders round a focus of dead caseous material from which the blood-stream is excluded, arrest and cure must be exceedingly slow. Consequently it is imperative in such cases to continue treatment for a very long time after the surface-lesions are healed. In a private patient one may get some information by an examination of the opsonic index before and after exercise in such cases, but an absence of auto-inoculation must be regarded with enlightened suspicion, since the lesion may be too removed from the blood-supply to furnish this test of activity.

A *fourth* point in this case is the appearance and continued presence of a tuberculous skin lesion while the patient is under tuberculin treatment. It reminds one of the fact, better exemplified in the second case I am going to show you, that tuberculin, though an invaluable remedy for tubercular lesions, yet confers no immunity against fresh infection. It also demonstrates how little good tuberculin, given in these small doses, accomplishes in tuberculosis of the skin. In my experience much may be done for this condition with large doses on the "intensive system," but very little by the small doses suitable for cases of localised tuberculosis elsewhere.

The second case is that of A. C—, æt. $2\frac{8}{12}$ years, who was admitted to the East London Hospital last September for operation on tuberculous neck glands. On his discharge he weighed 16 lb. and was put on bovine tuberculin T.R., and while having it developed a tubercular dactylitis of the right hand, which was opened in February. He was now given human tuberculin T.R. and the weight rose, but the hand did not heal on small doses. One thousandth milligramme (old nomenclature, better ·0001 c.c.) was now given at shorter intervals and the local lesion improved, though he lost weight and showed symptoms of intolerance. The dose was lowered, and he has continued to improve both in the healing process and the general nutrition, his weight having risen by 9 lb., *i. e.* by more than half his original weight, during the nine months he has been under treatment. At the present time he is having the bacillus emulsion (B.E.) in corresponding dosage, this preparation being some two and a half times as strong as tuberculin T.R., and the hand is quite healed.

His case exemplifies again the failure of tuberculin in establishing immunity to fresh outbreaks of tuberculosis. It also illustrates points not brought out by Case 1, and among them the seeming paradox that tuberculin in *large* doses cannot be given at *long* intervals. You will remember when doses are given at two or three days' interval, after the "intensive method," which we use with success in certain cases of phthisis, that here "*tolerance*" can be gradually established to very considerable doses. I find, however, that injections given at intervals of two weeks (the best interval in my experience in cases of localised tuberculosis) may lead to intolerance even with a moderate dosage, and if a large dosage is tried will almost certainly do so. This is probably an instance of the curious phenomenon known as "anaphylaxis" or hypersensitiveness, a phenomenon which is a very possible explanation of the "tuberculin reaction," and thus of the general symptoms of tuberculosis. When an animal is injected with a very small quantity of an "antigen" (egg-albumen will apparently suffice), *after an interval* of some days

it becomes hypersensitive to this antigen, and a further injection will lead to violent symptoms and often death. If the dose is repeated *before* "anaphylaxis" or hypersensitiveness appears nothing happens. Applied to tuberculin it will explain how "tolerance" can only be established by giving injections at *short* intervals (*i. e.* before anaphylaxis occurs), and how hypersensitiveness to quite moderate doses tends to arise when intervals of one or two weeks are left between the injections (anaphylaxis having time to appear in the interval). This is a theoretical consideration, but the fact which it seems to explain is of practical value (if others can support my experience), namely, that if *large* doses require to be given *they must be given at short intervals* if intolerance is to be avoided.

This case exemplifies another point, namely, the very, very slow effect tuberculin has on small, hard, unopened tuberculous glands. You will find some in the right side of the neck, and they haven't altered much as a result of treatment. In private more could have been done for these glands in the way of "irrigation" by the application locally of heat or of some suction apparatus. It must be remembered that the rational therapeutics of chronic infections must take two points into consideration: one is "irrigation" of the area with blood, and the other is the heightening in that blood of the supply of anti-bodies. Vaccination achieves the latter, but the former must not be neglected, and in some cases certainly the former is the more important measure of the two. The resistance of these glands to treatment exemplifies the probable condition of the tuberculous intrathoracic glands, which are almost certainly present in both these cases of localised tuberculosis.

The other cases I am showing you to-day exhibit the antithesis in tuberculous infection. They are cases of what one is tempted to call "ordinary" phthisis, save that no case of phthisis is "ordinary," each one being quite different and distinct from all others that have ever been seen or described. It is this protean aspect of pulmonary tuberculosis (more, perhaps, than of any other disease) which renders so futile all attempts at statistical inquiry as to its curability or recoverability. Each case stands by itself, each requires its own special handling, and it may fairly be said that what is food for one is poison for another. There is no royal road to treatment, but each case must be studied separately, due consideration being given to the all-important points: "amount of auto-inoculation," "amount of tolerance," "potential resistance," and a careful estimation of how far loss of tolerance, if such has occurred, is the result of advancing disease, how far of secondary infections, how far of temporarily lowered resistance. On account of the multiplicity of factors concerned in each single case of this disease it may safely be said that the "impressions" of experienced physicians, such as Trudeau, are worth all the statistics that will ever be compiled.

These cases of phthisis, as I have said, are the antithesis of the localised tuberculosis we have just considered. Here the disease is in full reach of the blood-stream, as is evidenced by the train of symptoms so familiar in active cases of this disease. One of these patients is an "early case," in that he complains of no cough and gives a history of only two or three months of lowered general health, but you will find the early signs in fully four lobes. The other is a case in the third stage, with marked loss of tolerance, and the symptoms of secondary infection of the diseased areas. I would invite you to consider not merely the "clinical signs" in these patients, but how you would treat them if they were handed over to your care for this purpose.

August 15th, 1910.

BERNHEIM claims for his treatment of breast infection that if the patient is seen early enough and pus-formation has not started, abscess formation can often be prevented by the institution of cupping for from fifteen to forty-five minutes daily, the breast between treatments being put up in a wet boric dressing, and nursing stopped. If an abscess has already formed, a wide incision is made, followed by drainage with iodoform gauze, and the cup is not used until twenty-four hours have elapsed. At the end of the cupping the whole breast is put up in wet boric gauze and very loosely bandaged. The advantages claimed for the method are almost absolute freedom from pain during the whole course of the disease, better preservation of the gland function, and the minimum of scar-formation.—*Med. Rec.*, vol. lxxviii, No. 3.

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CONTENTS.

PAGE

***A Lecture on Hysteria in Childhood.** By ROBERT HUTCHISON, M.D., F.R.C.P. 305

***With Mr. W. H. Battle in the Wards of St. Thomas's Hospital** ... 312

***The Practice of Inoculation of Smallpox.** By F. M. SANDWITH, M.D., F.R.C.P. 317

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A LECTURE

ON

HYSTERIA IN CHILDHOOD.

Delivered at the Hospital for Sick Children, Great Ormond Street.

By **ROBERT HUTCHISON, M.D., F.R.C.P.,**

Assistant Physician to the Hospital, and Physician to the London Hospital.

GENTLEMEN,—My subject this afternoon is hysteria in childhood, and you might perhaps expect that at the outset I should define what I understand by the term "hysteria." But in that I am afraid I must disappoint you, because, frankly, I do not know of any satisfactory definition. And if you attempt to penetrate into the inner meaning of hysteria you simply involve yourself in the fogs and bogs of a pseudo-psychology. That, at least, is the conclusion I have come to from a survey of the recent literature on the subject. I shall content myself, therefore, with a purely objective description based on my own clinical experience, and shall be satisfied if I convince you that hysteria is, indeed, a possibility to be reckoned with when one has to do with a case of obscure nervous disease in early life. Although I shall have many cases to relate, you must not make the mistake of supposing that hysteria is common in childhood. On the contrary it is rare, and I doubt whether it can be recognised with certainty below the age of five. At the outset certain points may be mentioned in which hysteria in childhood differs from the same condition in the grown-up person. One is with regard to sex distribution. There is not the same disproportionate occurrence of hysteria in the female sex in children that there is in adults. Indeed, I think you will find when I come to describe cases that it is quite as common in boys as it is in girls. But the nearer you get to puberty the more does the tendency for the female sex to

preponderate as subjects of the disease exist. Another difference which one notices between hysteria in children and in grown-up persons is that in children it tends to assume a simpler form. It is often what is termed "mono-symptomatic," that is to say, there is only one hysterical manifestation present. In that hysteria simply obeys the rule which I have pointed out in lectures here before, namely, that all disease processes in children tend to be simpler than in grown-up persons. And to that hysteria is no exception. Another point characteristic of hysteria in the child is that many of the so-called stigmata, such as anæsthesia of the pharynx, contraction of the visual fields, pressure points, and so on, either are not met with at all or only occur exceptionally. Why that should be I do not know, but it is a clinical fact which you must bear in mind.

With regard to the ætiology of hysteria in children, I think one can recognise with a fair degree of frequency two kinds of causes—(1) predisposing, (2) exciting. A large number of hysterical children are disposed by inheritance to suffer from the condition. You will be surprised how often, when you are dealing with an hysterical child, you will find that it has an hysterical mother or that it is the offspring of neurotic or highly emotional parents, and this predisposition has often been aggravated and fostered by the circumstances of the patient's environment. The child is apt to have been badly brought up, not in the sense of having been neglected—on the contrary it has probably been coddled and spoilt—but it has not been brought up to exercise self-control, and very often in an emotional and exciting atmosphere.

Of the exciting causes of hysteria in childhood the commonest is injury of some sort, physical injury or trauma. I shall give you some concrete examples of this immediately. The other great exciting cause is what has been called "psychical trauma," namely, fright, and in a number of instances hysterical manifestations have followed upon this. That is all I can say definitely from my own experience about ætiology, but in a great many cases you will be unable to find any cause for the condition at all.

Motor Manifestations.

Hysteria may assume various forms, the commonest, perhaps, being affections of the motor system, which may show themselves as paralysis, fits, spasms or tremors. And first let us speak of *hysterical paralysis*. Of the different forms of hysterical paralysis in children, monoplegias and paraplegias are the commonest. Hemiplegias are less frequent. The paralysis is apt to be of sudden onset, and it usually assumes the flaccid or limp type. Hence the case is apt to be mistaken for one of infantile paralysis, as happened in the following example:

Case 1.—A boy, æt. 12 years, was brought up from the country with a history of paralysis of the right arm, which had come on suddenly five months before. The paralysis had been regarded as due to poliomyelitis. On examination there was complete flaccid paralysis of the right arm, but with only a moderate amount of wasting. There was anæsthesia of "sleeve" distribution up to the shoulder. Otherwise he seemed normal. Under the application of faradism accompanied by vigorous suggestion he immediately recovered the use of the limb, and returned home next day cured.

I have not myself seen a case of hysterical paraplegia in a child, but those who are interested in the subject will find a very instructive example of it, which led to various errors in diagnosis, recorded by Dr. C. W. Chapman in the fifth volume of the 'Transactions of the Society for the Study of Disease in Children.'

Hemiplegias are comparatively rare, and when they do occur the face usually escapes. The following is an example:

Case 2.—Susan C—, æt. 12 years, got a severe fright at a fire two years ago, and had had several "hysterical" fits since. After one of these lately she lost the use of the right arm and leg. The arm quickly recovered, but the right leg remained weak, the foot being bent in the equino-varus position and dragged along as she walked.

Examination showed anæsthesia over the whole of the right half of the body, with amblyopia in the right eye and considerable areas of analgesia in the leg. Otherwise examination was negative, but her mother said that she would sometimes refuse all food for two days on end.

She only came once or twice to out-patients, and her subsequent history is unknown.

The curious condition called hysterical astasia-abasia—inability to stand or walk—seems also to occur sometimes in children, but not commonly. Usually I think it is left behind as the result of a condition which at the outset did really make standing or walking painful, and then after that has

passed off, the astasia-abasia persists owing to the hysterical tendency. This seemed to happen in the following case:

CASE 3.—Albert L—, æt. 5 years, "went off his legs" when eighteen months old (? result of scurvy), and had never walked since. He seemed quite healthy, there was no paralysis, and he kicked his legs about freely in bed. The knee-jerks were rather brisk and the plantars gave a flexor response. At first when one tried to make him stand he simply screamed and doubled up his legs, but after a few days of persuasion he stood up, and four days later was walking quite well.

There was a good deal of anorexia whilst he was in hospital, and it was difficult to induce him to take enough food.

This, I think, was a case which might be fairly described as hysterical astasia-abasia. It had started, I believe, owing to the tenderness of the limbs produced by scurvy, and afterwards the inability to walk was perpetuated in an hysterical form.

Passing from hysterical paralysis, we come to *hysterical fits*. They are, as you know, a very common manifestation of hysteria in the adult, the typical case being that of a young woman who gets an "hysterical attack" in which she throws herself about, cries, laughs, screams, etc. That type of hysterical fit is not common in children, although I shall have occasion later on to mention examples which might be referred to it. Much more commonly an hysterical fit in early life closely simulates epilepsy, and sometimes the simulation has been so close that it requires great care to distinguish one condition from the other. I can give you two remarkable examples:

CASE 4.—Arnold H—, æt. 14 years. About five weeks before he came under observation this boy fell down in the school playground in a "fit," from which he speedily recovered, but since that time he had been subject to right-sided convulsions, which were getting more frequent. For the last two days he had not spoken.

He had had an attack of rheumatic fever at the age of ten, which had left him with a mitral lesion, but otherwise he had enjoyed good health. His parents were both said to be "nervous." There were four other healthy children.

He was a bright, healthy-looking boy, but exhibited complete "mutism." The fits recurred at intervals of a few minutes, and consisted in slight twitching of the right arm, leg, and face. The attack began with a slight grunt and ended with a sigh. He was partially unconscious in the attack, but the conjunctival reflex was not entirely abolished. The eyes showed conjugate deviation in the attack, usually to the right, but sometimes to the left. The pupils were equal. He never bit his tongue nor passed water in a fit. There was slight weakness of the right arm, leg, and face; and the knee-jerk on the right side was not so easily obtained as on the left. There was no anæsthesia, and the fundi were healthy. He readily obeyed commands, but wrote with great difficulty, and seemed to forget words and how to spell. He did not appear able to read print.

Under isolation with the use of faradism, suggestion, and cold shower baths he quickly regained his speech, the fits stopped, and he left hospital quite well about three weeks after his admission.

CASE 5.—William W—, æt. 15 years, was seen in 1906 for "fits," which had begun three days previously. His mother had suffered from some sort of "fits," but patient himself had always been healthy. The fits in his case had begun with "stiffness of the jaw," after which he was said to have lost consciousness. The fits had since been repeated nearly every quarter of an hour. He had never bitten his tongue in them, but once at least he had passed water during an attack. For the last twenty-four hours he "had not spoken."

On examination he seemed placid and intelligent, obeying commands readily, but quite mute. When a fit came on the right side of the face twitched violently and the hands were clenched, but showed no clonic spasm. There was no loss of consciousness and the conjunctival reflex was not abolished. The pupils were dilated, the knee-jerks normal, and the fundi healthy. There was apparently no anæsthesia.

The fit lasted about a minute and was repeated at short intervals.

He was admitted to hospital, and after about ten days recovered his speech and the fits ceased.

In hysterical fits of this type I think mutism is a symptom of great diagnostic value. In both these cases its presence clinched the diagnosis.

Passing from fits, one comes to hysterical *contractures of joints*. These are rather common manifestations of hysteria in children, and I have seen several examples of them. They often follow injury of the joint, which has caused pain and led to some degree of flexion, and that, owing to the hysterical basis, has become perpetuated and exaggerated so that there is extreme spasm. One of the best examples I have seen was the following:

CASE 6.—Jane K—, æt. 11 years, was admitted to hospital for spasm of the hands of two to three days' duration. She had had several attacks of a similar sort before during the preceding year and the

feet also had been "doubled up," but the attacks had always passed off quickly in hospital. The patient herself said that the spasm began with a feeling of "giddiness"; then the thumbs bent in and the hands closed so that she could not open them. There was nothing noteworthy in the family history, and up till a year ago the patient had always enjoyed good health.

She was a well-developed girl, with a rather sullen expression. The fists were tightly closed, with the thumbs bent in and the arms bent across the chest. By gradual pressure the fingers could be unbent and straightened out, and then she could move them quite freely. The knee-jerks were exaggerated, but there was no ankle clonus. Sensation was normal and the viscera healthy. The visual fields were not tested.

The spasm quickly disappeared in hospital, but she had a typical attack of "globus" whilst under observation.

It is important in cases of hysterical contracture not to overlook the fact that there may be a real organic basis for the condition. A child, for instance, may have commencing tubercular disease of a joint, or there may be some other disease of it, but instead of responding with the ordinary stiffness and slight flexion there is this exaggerated response, and you must beware in such a case of attributing the whole condition to hysteria.

The last motor manifestation I have to mention is *hysterical tremor*. There are various forms of such tremor. You may have a true intention tremor, which comes on only on attempts to use the affected part. That happened in the case of a boy, æt. 10 years, who had tremor of the hand as soon as he tried to write, although he used it for other purposes quite well. Sometimes the tremor consists in a series of clonic movements confined to one group of muscles—a repeated clonic spasm. There is little doubt that the condition called paramyoclonus in children is hysterical, and I shall have occasion to describe to you immediately some cases affecting the respiratory system, which, I think, fall under this group. It is a characteristic feature of such hysterical spasms that they tend to stop during sleep.

Sensory Manifestations.

The next group of manifestations which one has to describe are the sensory ones. These, I think, are less common in children than the motor. There may be hemi-anæsthesia, as in the case of the girl I described (Case 2), or there may be anæsthesia of one limb of the sleeve type, as in the case of the boy with paralysis of the arm (Case 1). But in a considerable number of cases the hysteria is mono-symptomatic, and there is paralysis of sensation without paralysis of motion. Hysterical hyperæsthesia is said to be commoner than hysterical anæsthesia in children, but I have not seen many clear examples of it, except those which are associated with contractures of joints, some of which are extremely painful. It is particularly difficult to be sure of the existence of an hysterical hyperæsthesia, and to exclude the possibility of underlying true disease, but not long ago I saw a case which I think may, perhaps, come under that heading. Some of you saw it when Mr. Waugh was looking at it with me in the out-patient department. The patient was a little girl, who was sent up with the history that not long before she had had an attack of whooping-cough, and ever since she had complained of pain in the abdomen, which almost prevented her walking. When she tried to walk she did so in a curious, stiff, cautious manner, and said it hurt her. Mr Waugh and I examined her thoroughly from every point of view —abdomen, spine, etc.—but could not find any sign of organic disease, and we both noticed that after the examination was completed, and the child thought she was no longer under observation, she walked ever so much better. Mr. Waugh thought it was functional, and I think it is probable that it was a hysterical hyperæsthesia, but I should be very cautious in giving such an opinion unless one had had the case under observation for some time.

Respiratory Manifestations.

Passing from sensory manifestations, there is a large and interesting group of hysterical symptoms in children, which one may class as respiratory manifestations. Of course mutism and aphonia are, in a sense, affections of the respiratory system, and both are common signs of hysteria in childhood. I have already described examples of mutism, and I would only point out here that it illustrates what I shall speak of more fully when I come to the question of diagnosis, namely, the tendency of hysterical manifestations to assume an exaggerated form. Instead of having a mere difficulty in speaking, as in aphasia, there is complete dumbness, that is to say, inability to speak at all.

and it is this exaggeration of the disability which is so suggestive of hysteria. I would remind you also of another characteristic feature of cases of hysterical mutism and aphonia, namely, that the child is still able to cough, although he cannot talk. In true paralysis of the vocal cords that would be impossible. I shall have to refer again to this inconsistency of the symptoms in hysteria.

One sometimes meets with an *hysterical cough*. A good case of that kind was the following:

CASE 7.—A boy, æt. 15 years, was brought to me for a cough, which went on incessantly, wearing out the nerves of all the other people in the house. He had had a similar attack three years previously, which passed off in about a month. A year later he had another, lasting three weeks. Later on in that year he was hit on the head by a cricket ball, and a week afterwards complained of pain behind the right ear. Nothing was found, but he kept holding a handkerchief to it all the time till the pain passed off, in about four weeks. Next year he got a worse attack of cough than ever, accompanied by great tachypnœa (120 per minute). The cough was attended by some frothy expectoration, but there were no signs in the chest or throat and no rise of temperature. It is noteworthy that the cough ceased when he was asleep. This attack passed off entirely, and he returned to school and remained well till the beginning of the present year.

He was a pale, rather poorly nourished boy, with a constant noisy cough, repeated every few seconds.

The lungs were perfectly healthy. The throat showed the remains of embedded tonsils, and the vocal cords were somewhat reddened. Examination with a probe showed some tender spots on the pharynx, which seemed to be the starting-points of the cough. The tonsils were enucleated, and the day after he did not cough at all. The following day he coughed as before, but next day he was much better. He subsequently made a good recovery.

Sometimes there is an *hysterical cry* or repeated utterance:

CASE 8.—Constance B—, æt. 7 years, was brought to my out-patients' in January, 1901, with the complaint that "she made a curious noise," and had done so, more or less, for the last five months, but it was getting more frequent, and occurred now nearly every day, lasting for from one to four hours at a time. The attacks were accompanied by pain in the throat and "round the heart."

She was the only child of a very emotional mother, precocious and rather spoilt, but well developed and healthy looking. She had never had fits, but had sometimes had "inability to walk," and had fallen down on several occasions in the week before she came under observation.

During examination the cry came on; it took the form of a curious indrawing sort of crow, very difficult to describe, and was frequently repeated. Physical examination was entirely negative. She was admitted to hospital, but during her stay in the ward was perfectly normal.

The following was a case of a very similar sort:

CASE 9.—A girl, æt. 11 years, was seen in consultation in November, 1907. She was giving vent at short intervals to a sort of suppressed scream. This had been going on for six weeks, but it stopped during sleep and meals. She was very excitable and emotional, and easily burst into tears. The attack was said to have begun with pain in the tragus of one ear, passing round the helix. She was examined at the outset by an aurist, but nothing was found except slight redness of the drum, and the pain was supposed to be neuralgic, and various drugs tried without success. She was undoubtedly a spoilt child, but I could find no signs of any organic disease, and her previous health was good.

She recovered quickly when removed to a Home.

Another very interesting respiratory manifestation is *hysterical dyspnœa*, or tachypnœa—rapid and laboured breathing without apparent cause. I have seen several such cases, of which the following are good examples:

CASE 10.—Osmond F—, æt. 8 years, was sent up to hospital with a history of laboured breathing for eight weeks, following a series of "fits," which had been preceded by obstinate constipation. In the fits he was said to be unconscious and threw his arms about, but did not pass water. He had always been delicate and constipated, and had a very poor appetite, living chiefly on biscuits.

On examination he showed continuous panting respiration (thirty-six per minute), the inspirations being forced, but without involvement of the accessory muscles, nor was the diaphragm over-active. His breathing became worse when he was looked at. There were no signs of disease in the lungs nor elsewhere, but he exhibited some twitching of the nose and mouth. The knee-jerks were brisk and the fundi healthy, but there seemed to be some contraction of the visual fields. He was treated by faradism to the spine, and left hospital in a month much improved.

CASE 11.—Edward J—, æt. 15 years. Five days before he came under observation he got a

blow on the head, which almost stunned him; since then his breathing had been difficult. He had always been nervous, but otherwise his health had been good. He breathed in a panting fashion, with forced inspirations at the rate of thirty-six per minute; the alæ nasi were active. There was no dyspnœa when he was speaking, and it was noticed that he could hold his breath for a long time then. The dyspnœa was worse when he was looked at, but disappeared during sleep. There were no signs of disease in the lungs or elsewhere, and no other stigmata of hysteria. He remained in hospital two weeks, being treated by isolation, suggestion, and cold shower baths, with full ether narcosis on one occasion. He recovered sufficiently to be able to go to a convalescent home, but his subsequent history is unknown.

Ocular Manifestations.

Passing from these one might next mention some ocular manifestations of hysteria. The first is *blepharospasm*, which is certainly hysterical in some cases. It usually begins with a slight conjunctivitis or ulceration of the cornea, which causes ordinary blepharospasm, and then the condition becomes exaggerated and perpetuated. I remember seeing a little girl, æt. 7 years, who exhibited this symptom in an extreme degree, one eye being screwed up as tightly as possible. She had had a good deal of conjunctivitis for some time previously but it had now subsided, and under suggestion, telling her she could open her eye and encouraging her to do so in the out-patient room, she opened it, and the spasm did not return.

You also meet sometimes with an hysterical *ptosis*. I have seen it associated with a *monocular diplopia*, a very curious condition. One also gets, amongst the ocular manifestations, *limitation of the field of vision*, but this is less constant in hysterical children than it is in adults.

Visceral and Mental Manifestations.

The last group of symptoms which one may mention are the visceral and mental manifestations of hysteria. These are much more difficult to define and to describe in the child, because one gets on the confines of congenital moral deficiency, or of mere bad temper, and also on the borderland of neurasthenia, and the precise distinction of hysteria from these conditions becomes difficult. Take the following case, for instance:

Case 12.—Girl, æt. 5 years. Since two years old has been subject to "screaming fits," which come on with the slightest provocation, *e.g.* when asked to see a visitor or the doctor, or if the other children get something she wants. She throws herself down in the attack, and kicks and screams at the top of her voice. After the attack she is rather exhausted and dazed.

There was no family history of insanity, but the father was an extremely choleric and uncontrolled person. The patient had been late in walking and talking, but was said to be intellectually bright, although her attention easily flagged. Her doctor stated that she was physically sound, but I found it impossible to examine her, as she simply lay and screamed and kicked on the floor of my consulting room.

A year later the attacks were said to be getting fewer.

It is very difficult to place such a case. Was it due to mere bad temper, to some moral defect, or to hysteria? In its want of control it certainly corresponds to hysteria, but I should not like to be sure about it. In other cases the child malingers or concocts groundless tales. Or the condition takes the form of pure emotionalism, sometimes with a religious colouring, as in the following instance:

Case 13.—Boy, æt. 10 years. Had always been backward, and was obliged to go to a "special" school, but went on all right till seven weeks before he came under observation, when for a week his "arms and legs became rigid," and he would not speak nor eat. This passed off, and was succeeded by fits of violence, in which "he breaks and tears things" indiscriminately. Physically he seemed quite healthy and unusually well developed for his age, with a curiously deep voice, but mentally he was rather vacant and sullen. His father was of opinion that the services during Lent had upset him, as he always followed the Salvation Army, and would stand up at any time and preach or pray!

That is a case which in its history and symptoms certainly fits in with hysteria better than anything else. Sometimes hysterical children exhibit groundless fears or "phobies," but such cases are nearly allied to neurasthenia. I saw a little girl not long ago, for instance, who was panic-stricken at any sudden change in the weather. A thunderstorm particularly upset her, and every time one came on she would rush under the bed and hide herself. A rain-storm or a gale of wind had much the same effect. It is difficult to say whether or not such a case should be regarded as one of hysteria. Sometimes one meets with anorexia, and sometimes

also with hysterical vomiting, but both these manifestations are rare in childhood.

Diagnosis.

The first step in the diagnosis is to *remember the possibility of the occurrence of hysteria in childhood.* Many cases are overlooked because people do not realise that hysteria does occur in childhood or early life, and is not, as some are apt to suppose, a condition peculiar to the adult. Secondly, you have to remember that hysteria, not only in childhood, but in the adult, is always a thing *to be diagnosed with fear and trembling.* Mistakes are very apt to be made through overlooking the fact that there is some underlying organic condition present. You know that such a mistake is apt to be made in the case of young women suffering from early disseminated sclerosis, and it is also true of organic disease in children. *A propos* of this there is an interesting case recorded by Dr. Chaffey in the 'Reports of the Society for the Study of Disease in Children,' vol. iii. It is that of a boy who was believed to have hysterical anorexia from emotional disturbance on the death of a favourite sister, but who finally proved to have a tumour of the brain.

Thirdly, remember that *symptoms and signs which cannot be voluntarily produced or simulated are probably not hysterical.* Most hysterical manifestations can be simulated or produced by an effort of the will if necessary. Take, for instance, the case of mutism. Anybody can be mute if he or she wants to, whereas such signs as changes in the optic discs, abolition of the deep reflexes, or true clonus, cannot be simulated or voluntarily produced, and as a matter of fact such signs are never hysterical, and if they are present you should not diagnose the case as one of hysteria. Fourthly, *there is often something exaggerated or bizarre in hysterical manifestations*—what the French speak of as the "massivity" of the symptoms—which is of help in diagnosis. An hysterical pain, for instance, is always an extreme pain, hysterical paralysis is always absolute paralysis, an affection of speech amounts to complete mutism, hysterical anæsthesia is profound anæsthesia, and so on. It is the profundity of the symptoms which is suggestive of hysteria. Fifthly, *it is suggestive of hysteria when the effect is out of proportion to the cause,* as when a child, for instance, gets a slight injury to a joint and develops a violent contracture. Sixthly, *inconsistency of the symptoms* with themselves is suspicious of hysteria. In the case of astasia abasia, for instance, the child can kick its legs about in bed freely, although it cannot stand on them, as I have mentioned. It may be able to cough, but not to speak. Or the signs and symptoms may be inconsistent with anatomical facts. A paralysis, for example, has not an ordinary nerve or centre distribution at all, or an anæsthesia may not correspond to any distribution of peripheral nerves, or to central nerve areas, as, for example, in the sleeve type of anæsthesia. This is absolutely conclusive of hysteria. Seventhly, *a sudden onset or disappearance of the symptoms or causeless variations in their intensity* is suggestive of hysteria. The symptoms due to organic disease, as you know, usually come on gradually, and, as a rule, they do not go away suddenly. Finally, *the maintenance of a good state of the general health,* notwithstanding the severity of the local symptoms, is suggestive. The two boys with hysterical fits, whose cases I described to you (Cases 4 and 5), were examples of this. As soon as the fit passed off they were well, there was no elevation of temperature or interference with the general health, which would have been the case if they had had a succession of epileptic fits.

Prognosis.

With regard to prognosis, all I will say is that the prognosis of hysteria in childhood is good as regards immediate cure, certainly better than it is in the adult, but the difficulty is to prevent relapses, because of the patient's inborn constitution and often because of his environment, and it may be part of your duty to change the surroundings if you can by sending the child away from home.

Treatment.

The first and most essential point in the treatment of hysteria in childhood is *isolation.* Get the patient into a home or hospital. Isolation is important because it takes the child out of the unhealthy environment, in which it has often been living, and that alone may be sufficient to effect a cure, as in some of the instances I have mentioned. Next, ignore, as far as you can, the condition of the child. Repeated examinations, demonstrations to students, and so forth, are bad for the patient.

Wholesome neglect is the best attitude to adopt, and certainly the doctor and nurse should not be over-sympathetic, though of course the child should be treated with the glove of silk on the iron hand. In dealing with specific symptoms, what has been called the "method of surprise" is good, that is to say, taking the patient unawares. In a case of astasia abasia, for instance, put the child suddenly on its feet and tell it to walk. Sometimes under those circumstances the patient is taken aback and does walk, and then you have no trouble with it afterwards. But the objection to that method is the risk that if the child be not successfully taken by surprise you have not only failed to effect an immediate cure but you have done something to perpetuate that symptom, and to make any subsequent treatment more difficult. But the manœuvre often comes off. "Suggestion" is of great help, suggesting, for instance, that the child can move the limb, or that the pain has gone away, or that it can straighten out an affected joint, etc. With regard to other aids, I still believe in the old-fashioned treatment by cold shower baths. Drugs, such as valerian, are perhaps helpful, and in some cases tonics may be necessary, iron, arsenic, and strychnine, so as to get the general health into good condition. It is extremely important also to see that the convalescence is made complete, and that the child is kept away from home for a considerable time until it is well out of the hysterical habit, as otherwise there is apt to be a relapse.

August 22nd, 1910.

WITH MR. W. H. BATTLE IN THE WARDS OF ST. THOMAS'S HOSPITAL.

HERE is a woman, æt. 27 years. She complains of a sore place in the neck. She says she was all right until three months ago, when the sore place came, but she had had a lump in the same situation for six years. It was not painful, but only itched, and she has not had any pain in it since it broke. She also has some scars of previous ulcerations on her legs, which were bad three years ago. These marks on the legs, you will observe, possess no particular characters now. They are irregular and superficial, are not pigmented, and have no definite outline. What do you think the place in the neck may be? You would describe it as a granulating sore over the middle of the clavicle, near the insertion of the sterno-cleido-mastoid. The actual granulating surface is not larger than a sixpence. In all ulcers you want to take note of several points in addition to the condition of the surrounding parts, the appearance of the surface, the base, the amount and kind of discharge, the condition of the edges, and the question of pain. This sore you would describe as nearly clean; there is only a little discharge. The granulations are a little above the level of the edges of the skin, and pale in colour. The ulcer is generally below the surface of the skin, and has bluish, rather undermined edges. The base is thickened, and attached to the clavicle, that is to say, the ulcer does not move over the clavicle. The condition of the parts round about may be described as satisfactory. You of course examine the bone with a probe to see if it is diseased. But inability to get to the bone by a probe does not necessarily mean that the bone is not diseased: you may not be probing in the right place. In cases of doubt you would be careful to take an X-ray photograph of the case. One has been taken here: there is no disease of the bone. In radiography they sometimes get shadows which are not an indication of disease. Remember radiography is not an absolutely perfect science; the result of the process is a shadowgraph, not a photograph. It might of course be a tuberculous condition, a deposit of tubercle which has broken down. What makes you think it might be tuberculous? It is very chronic, and has taken a long time to partly heal up. The congested purple edge of the skin around it is an important point, and the ulcer is adherent to the parts beneath. As you suggest, scars on the leg might be due to old gummata, and the question of syphilis has to be considered. But remember that gummata of the skin are circular, or have a tendency to take the circular form, and are distinctly pigmented, and if you find this appearance you have something to go upon. She has superficial ulceration which remains chronic, but apparently causes her very little trouble. She does not seek advice on account of the state of her legs. Is it congenital syphilis? Her teeth are good and sound and properly shaped. In the so-called Hutchinson's teeth of congenital syphilis the central upper incisors are pegged, notched, and of bad colour. And it is too old-standing for gumma. Gumma does not often get well of itself; it has a tendency to spread, although slowly. Few gummata remain six years without breaking down. But tuberculous

nodules sometimes do, especially if they undergo caseation. True, this patient has no enlarged glands, but the characteristics of the sore are so well marked that you cannot have any hesitation in saying it is tuberculous. It should be scraped, to get rid of the diseased granulation-tissue. Of course if X rays or radium were applied the patient would get well in a few weeks. In the same way people have the Finsen light treatment for tuberculous ulceration and get well under it; still, under other treatment they also get well. But no treatment invariably prevents breaking down again later on, and you cannot be certain it will not break down again even if it heals after it has been scraped. The tubercle in her case is but a local affection; she is well nourished, and there are no signs of tubercle about her chest.

This little girl, æt. 8 years, is the subject of surgical emphysema of the abdominal wall, chiefly on the right side, where crackling can be felt. She has had an injury to the chest, and escape of air followed on that. This emphysema never extends lower down the thighs than the deep layer of the superficial fascia. The air may go into the scrotum in the male. The general swelling may become so extensive as to cause the patient to get very large, I have only seen this extreme condition once, but it was so severe that the patient resembled an air-cushion more than anything else, and the note was tympanitic. It was the case of a small boy, who had been run over, with the result that several of his ribs were broken. You could put your finger on the abdominal wall and press, when the skin of the abdominal wall gave under the finger one to one and a half inches in some places before the muscle was touched. What should we do for such a condition? We must treat the injury, and let the emphysema alone. Surgical emphysema is not dangerous; it may extend into the face, closing the eyes, causing the patient to have a blank face like that of a dummy, all expression being lost. Sometimes these people cannot see for days. This little girl was playing with her little brother, who used her as a pin-cushion, pricking her with a needle, leaving the needle, which was a large one, inside, and there is a tender place below the angle of the right scapula where it could be felt under the skin, and from which it was easily removed by incision; it penetrated the lung. A fractured rib may tear through the parietal and visceral layers of the pleura. Why does the air not go mostly into the cavity of the pleura instead of coming outside? The majority of cases of emphysema which are admitted after fractured rib have shown no disease of the pleura. Why does the air come through on to the surface of the body? It may go into the pleura as well, but it is not common to find that of importance in any great proportion of cases. There is a natural close cohesion between the two layers. The power of this resistance is usually greater than the force of the air attempting to overcome it, unless of course the air is violently forced in. Occasionally the lung is held by the fractured end after injury, and occasionally there has been present an old inflammation which has held the surfaces in contact. But other things besides fractured rib will cause surgical emphysema: you may find it as a result of fracture of the clavicle, and of wound of the trachea. It may be caused by over-exertion. It may result from the straining of parturition, or at stool; the trachea may give way and there may be emphysema of the neck, which may spread a long way. As a result of ulceration at the root of the lung there may be a giving way into the mediastinum, from which the air passes along the vessels to the neck. Surgical emphysema is occasionally associated with injury to the bones of the nose, and the patient gets an extravasation of air into the orbit and about the face should he inadvertently attempt to blow his nose.

This patient is æt. 37 years. She is now in the fourth month of her fifth pregnancy. On June 14th she had severe abdominal pain, and she came in on the 19th. The case is important in regard to the relationship of appendicitis to pregnancy. She has never had an attack of this kind of pain before. She vomited once, and the pain subsided. The bowels were opened once, on June 15th. She got up on Friday, and on Sunday night she had a very severe attack of abdominal pain, which was even worse than the previous attack. She again vomited. The bowels were not opened. There was no pain on micturition. The pain was severe, and distributed over the whole abdomen; it had never been localised to one spot. In her four previous pregnancies she had had no trouble. The importance of that is this, that the patient is unlikely to have old disease about the appendix, and that is very important from the point of view

not only of prognosis but from the question of operation. I am talking of ordinary appendicitis in pregnant women. If a patient has had recurring attacks of appendicitis she is not unlikely to have had adhesions in the neighbourhood of the appendix, perhaps binding the appendix and the omentum down, with the tube and ovary, and perhaps the uterus. And as soon as the uterus enlarges these parts get pulled upon, and there is pain, and that pain may terminate in an attack of inflammation of the appendix. This is very important, because the symptoms are not likely to improve as the pregnancy progresses. The patient is likely to have pain for some time, and then perhaps the formation of an abscess. And if you have a history of the patient having recurrent appendicitis and the uterus begins to get large, you ought to operate early so as to stop the pain as well as to get rid of the danger from the diseased appendix. Some people go so far as to tell you, and I think rightly, too, that if you want a patient to be safe you should always remove the appendix before anything of the kind is likely to occur, that the dangers attending a diseased appendix under such circumstances are so great that you should always remove the appendix before pregnancy occurs, or before there is a chance of its occurring in married life, or failing that, do it as soon as the symptoms arise. This patient had a very much worse condition than ordinary appendicitis. She came in with a history a little longer than usual. When she entered the hospital she had an excessive abdominal tenderness, a pulse of 108, and temperature 98·6° F. There was sharp superficial tenderness, no localised dulness or swelling, and the patient looked ill. We concluded she had a commencing peritonitis, and I operated as soon as possible, removed a gangrenous appendix, and found that the gangrenous condition had spread to the cæcum. The piece of the cæcum which was gangrenous was excised and the opening closed with a continuous stitch, and two lines of peritoneum were sutured over that so as to make certain it was closed in thoroughly. The cæcum around was œdematous, the appendix adherent to the surrounding parts, whilst the wall of the uterus on the right side and the ovary on the right side, and tube, too, were œdematous. So there was a fairly acute inflammatory condition, and in addition there was a quantity of offensive pus effused, principally in the right flank towards the right kidney. The pelvis was more or less filled up by the enlarging uterus. Douglas's pouch contained very little pus. I put a drainage-tube in afterwards, and the patient is recovering very satisfactorily. But acute appenditicis attending such a case as this is sometimes very fatal if you do not operate early and stop the inflammation. What happens is that there is an extension to the uterus, the uterus expels its contents, and you get a diffuse peritonitis and a septic condition of uterus, which in many cases has terminated fatally. The diagnosis of ordinary appendicitis in pregnancy is not always easy, for the simple reason that any pain which there may be is put down to a possible tendency to abortion or miscarriage. The doctor sees the patient, and she says, "I have some pain here and tenderness in the side." He asks her if she has been sick, and she says "Yes." He then says to her, "You must be quiet, or else you will have a miscarriage." Probably he does not think of appendicitis at all, and perhaps the condition goes on to abscess or something worse before he has made up his mind what it is. If, with those symptoms, you find the patient has a rise in temperature, you should suspect the possibility of appendicitis. There is an agreement amongst surgeons who have had most to do with the appendix that it is advisable to get rid of the appendix as soon as possible in these cases. Most cases will not miscarry if operation is done, and you should advise the patient to get rid of it, because it is the safer course, yet the attack will occasionally pass off without serious consequences. Still, the risks are so much greater in leaving it that you should advise removal of the appendix. Here I put in a tube up towards the kidney pouch, so as to make certain that there was no chance of pus accumulating afterwards. In the case where there is distinct localised abscess forming, you put in a tube in the same way. You cannot afford to search for the appendix if there is a localised abscess, or do anything to prolong the operation. There are cases in which this has been quite successful. The earlier in pregnancy you treat such a case as this, the better is the prognosis. In any case of pregnancy pus about the abdomen is very serious indeed. Another point which you have to consider is the onset of the

peritonitis at the time of the confinement. If a patient has an appendix inflammation and miscarriage takes place, or there is confinement at full term, it is very likely there will be rupture of an abscess into the general peritoneal cavity. And if you are not on the lookout you miss the fact that the patient has pus about the appendix, and the illness is put down as due to a septic inflammation of the uterus. There are many cases which have been treated wrongly because the uterus was only secondarily septic. It has been scraped, washed out, and plugged with the idea of stopping the inflammation arising in it, whereas the mischief has been in the peritoneum outside. There have also been many cases in which the exact condition of things was only found post-mortem. We are lucky in having residents who know their work, for it resulted in recognition of the urgency of this case, and the patient being operated upon immediately. Her condition was a very bad one. Gangrene of the appendix is the worst form of disease of the appendix you can have, and I only wonder that the patient has not had more serious symptoms. The tenderness was very excessive, and in all abdominal cases where there is excessive tenderness on the surface of the skin, and the patient jumps when you touch her, it means there is a spreading inflammation in the neighbourhood. The uterus was only half way to the umbilicus, and the state on percussion very little changed. I have seen this acute sensitiveness to touch in cases of perforated intestinal ulcer where there has been a margin of spreading peritonitis, and where other symptoms seemed inadequate to direct one to operate. It is seen also in acute hæmorrhagic pancreatitis, and I have no doubt it is a very important sign. I am always suspicious when it is found. Not long ago I saw a case in private in which a man had refused to have an operation in the quiet interval. He was æt. 50 years, and at that age it is more serious than usual to have these attacks, because they are sometimes rapidly fatal. A man at fifty does not stand such an attack like a man between twenty and thirty years of age. This man had had two or three attacks, and I had advised him to have his appendix removed in the quiescent period. But he had put it off. I was called in one Saturday, the illness having commenced two days before. He did not seem to be suffering much, but there was that excessive tenderness about which I have spoken as indicating the margin of a spreading peritoneal inflammation. I operated at once, and found pus free in the peritoneal cavity and a perforated appendix, with numerous adhesions about the appendix.

This woman is æt. 57 years. She came in for a condition of the pudenda, which she has had twelve months. She complains now of much burning pain. At the vulva you see a hard, thickened ridge, with a tendency for the outer edge to evert. There are some hard granulatious on the surface, and it is extremely sensitive. She has had a good deal of treatment, but nothing has done it any good. There is no particular involvement of her general health. The glands are not enlarged, and nothing else has been made out beyond what you have seen. What is it? Is there any reason why a person of her age should have such a condition? Remember it is very hard and definite—a chronic inflammatory condition would not limit itself to such a definite outline. Here is a swelling which comes from the clitoris and reaches two inches down the labium minor on the left side, and on the right labium minor for three quarters of an inch. The skin, mucous membrane, and subcutaneous tissues are infiltrated and it is excessively hard, with the outer margin turned outwards. A few glands can be felt in the groin, but they are not characteristic of new growth. The condition has been gradually extending during the past twelve months. It has, of course, been an epithelioma from the commencement. What can we do for it? We must remove the clitoris, labia minora, and glands, taking out a Λ-shaped piece, carry the incision well down to the pubes, and extend the lower part down to the under margin of the ramus. The piece removed must be very wide in its apex. Lymphatics in this region are very numerous, and recurrence is accordingly very likely. Later, if not removed, these conditions get very painful, and the patient suffers much on micturition; later still it may involve the orifice of the urethra. The condition may be so bad in neglected or recurrent cases that you are justified in doing what I have had to do where the urethra has been very much affected—take away the whole urethra down to the bladder, close the bladder below, and make a supra-pubic opening. I have done it three times with relief to the patient.

This man is æt. 30 years, and his trade is that of a carpenter. His right hand is perfect, but his left hand, from the little and ring fingers into the palm, is in a hard and contracted state, so that he cannot extend the fingers on the hand; in other words, he has Dupuytren's contraction. It is not due to manual work, because it often occurs in people who do not use their hands for anything in the way of rough work. It usually affects both hands. Here is a case in which it is only in the left hand; certainly, if there was anything in the theory of causation by work it should be the right hand which should be affected, for the man is not left-handed. The palmar fascia between the little and ring fingers is affected. There is nothing else which looks abnormal. There is a lump caused by very excessive thickening underneath the fat of the palm, and there is another lump, which is on the band of fascia going towards the thumb. That, again, shows you that it is not due to pressure. Most people think it a gouty affection, whilst others hold that it is due to rheumatoid arthritis. You cannot, of course, always prove it. Some say a man with this condition has the gouty diathesis; they say the fact of his having Dupuytren's contraction proves this, although he has never had gout. The late Mr. Anderson's idea was that it was probably due to the entrance of some bacillus through the skin. On closing the hand tightly the finger-nails dig into the palm of the hand nearer these two fingers than further on, and the disease starts where the injury of the nails would be most marked. He thought that this bacillus had the power of producing a chronic inflammatory change, but not suppuration. I do not know whether Mr. Anderson found the bacillus, but he had the fascia examined. He further pointed out that it is never met with in the plantar fascia, and that it should be if of constitutional origin. Of course there may be something in that. The skin is not very well supplied with blood-vessels, and it is very adherent to the fascia. There is some fear of sloughing of the skin after operation on these cases; it has been known to happen, and stitches often cut out. In doing the open operation, remember that flaps are not of very great vitality, and may slough. Fibrolysin has been used for the condition, but I do not know that many people are greatly in favour of it. There have been cases recorded in which purpura hæmorrhagica has developed during treatment with it. This statement is apt to act as a deterrent if you would use a remedy for a condition which is not of itself likely to prove fatal. Mr. Clutton told me he had tried it in many cases and found it of no use. Do you know Adams's operation? It consists of multiple subcutaneous punctures of the palmar fascia. In doing this operation be careful to cut the contraction furthest away from the fingers first, and then put the hand up antiseptically, allowing eight or nine days for it to heal. The patient must try to exercise the fingers after the operation. Afterwards he should put his hand into warm water and massage the fingers carefully so as to maintain the benefit given by the operation. He should wear a splint at night.

Here is a man with a fluctuating swelling, the size of a Brazil nut, on the right side of his neck, which he has had two years. It is not painful, but it varies in size. He notices that it is bigger after some meals than at other times, but is not very certain of this. It is larger one day than another, so the patient tells us. There is no pain. He has sometimes rubbed it, and that makes it swell a little. It might be a cyst of the parotid gland, or it might be a swelling secondary to a stone in the duct. There is, however, no soreness or swelling about the duct, nor is there any enlargement of the parotid. It is very rare to get a cyst about the parotid at all. Sometimes there is a dermoid cyst in this situation. Cysts of the parotid are usually cysts which develop in connection with tumours. This is a soft swelling, freely fluctuating, of distinctive outline, situated between the angle of the jaw and the sterno-mastoid. It runs down the neck for some distance, and does not encroach on the surface of the bone. It fits in between the parotid and the angle of the jaw. The lobule of the ear is not lifted, and there is no swelling there. If there is an enlargement of the parotid you expect to see the lobule of the ear lifted. We may dismiss the question of dermoid. It is not of sufficiently long duration. The fibrous structure of the parotid is very dense. He can open his mouth easily without any stiffness. Why should not it be a suppurating gland? There is usually more than one gland affected. It is characteristic of a tuberculous condition, that it takes a long time to develop. I remember seeing a case which which was sent over from Canada, to be under the care of Sir William MacCormac. The man had a tumour over his clavicle, which he had noticed for three or four years, and it had been named "cystic hygroma." It was simply a suppurating gland, the greater part of the glandular tissue having been absorbed, the capsule remaining, and filled with thin purulent fluid. It is true there is not anything here in the way of inflammation, but this swelling does not run into the parotid; it is below the parotid. I think this condition before you is most likely a suppurating gland. It is under the deep fascia of the neck. He is quite well except for this condition.

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THE PRACTICE OF INOCULATION OF SMALLPOX.*

By F. M. SANDWITH, M.D., F.R.C.P.,
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JOHN EVELYN, the celebrated author and diarist of the seventeenth century, at the age of twenty-six, while riding across the Alps on his return to England from Venice was struck down with smallpox. He arrived one night at Beveretta in the Duchy of Savoy, and says: "Being extremely weary and complaining of my head, and finding little accommodation in the house, I caused one of our hostess's daughters to be removed out of her bed and went immediately into it, whilst it was yet warm, being so heavy with pain and drowsiness that I would not stay to have the sheets changed; but I shortly after paid dearly for my impatience, falling sick of the smallpox so soon as I came to Geneva, for by the smell of frankincense and the tale the good woman told me of her daughter having had an ague, I afterwards concluded she had been newly recovered of the smallpox." Soon after reaching Geneva he felt as if his very eyes would have dropped out, and such a stinging about him that he could not sleep. He was attended by a very learned old physician, who at once bled him, purged him and applied leeches, and would have bled him again had not the smallpox eruption appeared. He was then kept warm in bed for sixteen days, and was nursed by a vigilant Swiss matron with a monstrous goitre, which used to frighten him when he awoke from uneasy slumber. The eruption was not extensive, and he lost his pain, though he was "infinitely afflicted with heat and noysomeness." "By God's mercy after five weeks' keeping my chamber I went abroad."

This gives us a good idea of the treatment patients received in those days. In England, Dr. Sydenham (1624–1689) made a very special study of smallpox, with the result that he advised a totally different mode of treatment, which he carried out with apparent success on his own patients. He made an important distinction between discrete and confluent attacks, and in the former case he left Nature to fight her own battle, recommending the patient not to keep his bed at all; even at the height of the illness he was to get up for several hours each day, "for whoever labours under the distinct kind," he says, "hardly needs the aid of a physician, but gets well of himself and by the strength of his nature." When we compare this treatment with Evelyn's account we appreciate the good common sense of the man who recommended it. If, however, about the third day, the smallpox threatened to be of the confluent type, Sydenham's treatment became more heroic and he advised active interference. Dr. Dover, who was a disciple of Sydenham, gives us an account of his master's methods.

"Whilst I lived with Dr. Sydenham," he says, "I had myself the smallpox and fell ill on the twelfth day. In the beginning I lost twenty ounces of blood. He gave me a vomit. I went abroad, by his direction, till I was blind, and then took to my bed. I had no fire allowed in my room, my windows were constantly open, my bedclothes were ordered to be laid no higher than my waist. He made me take twelve bottles of small beer, acidulated with spirit of vitriol, every twenty-four hours. I had of this anomalous kind to a very great degree yet never lost my senses one moment." The system of bleeding the patient at the onset of the disease, which Sydenham recommended even for infants and young children, was with the hope of checking the ebullition of the blood and so lessening the violence of the disease. We should now consider that such a weakening treatment could only increase the severity of the attack. When the patient was convalescing Sydenham recommended cordials and canary wine.

Bleeding in smallpox was very generally practised in France, where, it is said, it was done even to sucklings, but many disapproved of it; also Sydenham's cooling regimen was considered by many to be disastrous.

In 1718 Dr. Woodward, Gresham Professor of Physic, published some remarks directed against Dr. Mead and Dr. Friend, two upholders of the system of bleeding. Several abusive pamphlets were circulated on the subject, and one day Mead and Woodward met accidentally under the gate of Gresham College, where the professors of that day resided, and they instantly fell to fighting with their swords. We have only Woodward's account of the affair; perhaps Mead's version of it would be different. Woodward says that an accidental fall

* A lecture delivered at Gresham College.

during the duel put him at Mead's mercy, when he was driving Mead before him. Afterwards Mead upbraided him with not having asked his life; Woodward replied that Mead had shown himself a coward, and added: "That had he been to have given me any of his physic, I would rather than take it have asked my life of him; but for his sword, it was very harmless; and I was ever far from being in any the least apprehension of it."

Dr. Morton, who practised at this period, divides the cases of smallpox into three kinds, the benign, the medium, and the malignant, and he gives a long list of causes which may result in a benign case becoming a medium or malignant one. Some of these causes may here be quoted:

"If the attack fall in the flower of life, when the spirits are keener and more inclined to febrile heats. If the patient be harassed by fever, or by sorrow, love, or any other passion of the mind. If the patient be given to spirituous liquors, vehement exercise or anything else of the kind that tends to irritate spirits. If the attack happen in summer. If the patient be apprehensive of the result."

At about this time Dr. Woodward received a communication from a Dr. Timoni, a Greek living in Constantinople, who brought to his attention the practice of inoculation for smallpox, which, as he told him, was much in vogue in that city. This practice was to introduce the matter from a smallpox pustule of a discrete type into the tissues of a healthy person, in order to bring about an attack of the disease at a convenient moment and to render him immune for the rest of his life. The attack thus given was generally a mild one, and as the patient was almost inevitably doomed to take the smallpox at some time of his life the practice was found convenient, and was certainly not considered unjustifiable.

This inoculation is said to have been carried on in China a thousand years or more B.C. In India the custom is, perhaps, still older, and in spite of the efforts of a more enlightened age inoculation is still carried on in some parts of both these countries, in Tunisia, Persia, and elsewhere. From the far Eastern countries it was introduced into Europe, and the attention of several European doctors was directed to it.

It so happened that the English Ambassador in Constantinople at this time was Mr. Wortley Montagu, whose brilliant and vivacious wife was Lady Mary Wortley Montagu. She was the daughter of Lord Pierrepont, afterwards Duke of Kingston, and she married in 1712. On the accession of George I, Mr. Montagu obtained a seat on the Treasury Board and the young couple lived in London, where Lady Mary soon gained a reputation for talent and beauty, and was on terms of intimate friendship with Pope and Addison and all the wits of the day. In 1716 Mr. Montagu was appointed Ambassador to the Porte, and he went to reside at Constantinople with his wife and son. During her life in the East Lady Mary kept up a constant correspondence with her English friends, and one of these letters, written in 1717, I will now read to you.

"*Apropos* of distempers, I am going to tell you a thing that I am sure will make you wish yourself here. The smallpox, so fatal, and so general among us, is here entirely harmless by the invention of ingrafting, which is the term they give it. There is a set of old women who make it their business to perform the operation every autumn, in the month of September, when the great heat is abated. People send to one another to know if any of their party has a mind to have the smallpox; they make parties for this purpose, and when they are met (commonly fifteen or sixteen together) the old woman comes with a nutshellful of the matter of the best sort of smallpox and asks what veins you please to have opened. She immediately rips open that you offer her with a large needle (which gives you no more pain than a common scratch), and puts into the vein as much venom as can lie upon the head of her needle, and after binds up the little wound with a hollow bit of shell. . . . The children or young patients play together all the rest of the day, and are in perfect health till the eighth. Then the fever begins to seize them, and they keep their beds two days, very seldom three. They have rarely above twenty or thirty in their face, which never mark, and in eight days' time they are as well as before their illness. Where they are wounded there remain running sores during the distemper, which I don't doubt is a great relief to it. Every year thousands undergo this operation, and the French Ambassador says pleasantly that they take the smallpox here by way of diversion, as they take the waters in other countries. There is no example of anyone who has died in it, and you may believe

that I am very well satisfied of the safety of the experiment, since I intend to try it on my dear little son.

"I am patriot enough to take pains to bring this useful invention into fashion in England, and I should not fail to write to some of our doctors very particularly about it if I knew any one of them that I thought had virtue enough to destroy such a considerable branch of their revenue for the good of mankind. But that distemper is too beneficial to them not to expose to all their resentment the hardy wight that should undertake to put an end to it. Perhaps, if I live to return, I may, however, have courage to war with them."

True to her faith in the practice, Lady Mary had her son inoculated in the following year, and wrote: "The boy was engrafted last Tuesday and is at this time singing and playing and very impatient for his supper. I pray God my next may give as good an account of him. I cannot engraft the girl; her nurse has not had the smallpox." Mr. Wortley was recalled to England in 1717, and the following summer they returned to London. Faithful to her patriotic resolve, Lady Mary endeavoured to introduce the practice of inoculation into her own country, but she had prophesied with truth when she foretold that she would encounter a storm of opposition. "The faculty rose in arms to a man," she says, "foretelling failure and the most disastrous consequence; the clergy discanted from their pulpits on the impiety of thus seeking to take events out of the hand of Providence, and the common people were taught to hoot at an unnatural mother who had risked the lives of her own children."

The Wortleys' son had been inoculated in Turkey, but their daughter underwent the treatment after their return to England. The Government appointed four leading physicians to watch the case, but they were so sceptical, according to Lady Mary's account, so anxious *not* to see it succeed, that she dared not leave the child alone with them. The little girl sickened on the tenth day, the smallpox appeared, and she escaped with a very mild attack.

Caroline, Princess of Wales, who was on intimate terms with Lady Mary, now wished to have her children inoculated. Before doing so she proposed to the King (George I) that he should remit the capital sentence on six criminals in Newgate on condition that they should submit to the operation as an experiment. The King consulted Sir Hans Sloane, the well-known physician, who again applied to Dr. Terry, who had lived in Constantinople. His report of inoculation being satisfactory, the King gave his consent, and the six felons were duly inoculated. In each case the result was a slight smallpox eruption only, and they won their life and liberty. But this experiment alone did not satisfy the Princess, who next procured six charity children from the parish of St. James's and had them inoculated. Four of these had smallpox very favourably, another escaped, and the sixth is said to have had an attack of inoculated smallpox and an attack of the natural kind eleven weeks later. After this five more hospital children were inoculated, two of whom had mild attacks, whilst three escaped. Then at last the Princess ventured to have two of her own daughters inoculated, under the direction of Sir Hans Sloane. It is said that she was finally persuaded by the fact that another daughter had just had a violent attack of the smallpox, from which she narrowly escaped with her life.

The successful inoculation of the Princesses converted the Princess of Wales into a powerful ally of Lady Mary, and the practice came into vogue, somewhat in spite of the medical profession. Lady Mary's numerous friends consulted her on the subject, begged for guidance and tormented the poor lady for advice.

"Lady Byng," she writes, "hath inoculated both her children; the operation is not over, but I believe they will do very well. Since that experiment has not yet had any ill effect the whole town are doing the same thing, and I am so much pulled about, and solicited to visit people, that I am forced to run into the country to hide myself." Among her bitter opponents in later life was Horace Walpole, yet he had himself inoculated at this period, and therefore owed her some gratitude. In a letter Lady Mary announces the death of a little nephew from smallpox, and adds: "I think" the mother "has a great deal of reason to regret it, in consideration of the offer I made her two years together of taking the child home to my house, where I would have inoculated him with the same care and safety I did my own. I know nobody that has hitherto repented the operation; though it has been very troublesome to some fools, who had rather be sick by the doctor's prescription, than in health in rebellion to the college."

But the enthusiasm of the public received an early check when the young son of the Earl of Sunderland died of inoculated smallpox, and other similar cases came to be known.

But in spite of this it was in process of time very generally adopted and grew in popularity.

It was considered so inevitable a phase of life to contract smallpox that in those times the poorer people of England seem to have taken no precautions to avoid the disease; on the contrary, they exposed their children to the infection of mild cases, believing that this led to their taking the disease in a favourable form. We know now that this belief is quite erroneous, for from a mild attack may be caught one of extreme severity. A surgeon of Upton, writing in the latter part of the eighteenth century, says of the country people in his neighbourhood: "They neither feared it nor shunned it. Much more frequently, by voluntary and intentional intercourse, they endeavoured to catch the infection." Prof. Monro, of Edinburgh, wrote in 1765: "When the smallpox appears favourable in one child of a family, the parents generally allow commerce with their other children with the one in the disease; nay, I am assured that in some of the remote highland parts of this country it has been an old practice of parents whose children have not had the smallpox to watch for an opportunity of some child having a good, mild smallpox, that they may communicate the disease to their own children by making them bedfellows to those in it, and by tying worsted threads wet with the pocky matter round their wrists."

Although inoculation was adopted by the Court, by Court doctors, by the Royal Society, and by many of the leading practitioners, there were many others, chiefly physicians and clergymen, who were much opposed to it.

The clergy considered that the visitation of an all-wise Providence should not be interfered with. A sermon still exists against the dangerous and sinful practice of inoculation, preached by the Rev. Edmund Massey in 1722, in which he asserts that "The Devil, by some venomous infusion into the body of Job, might raise his blood to such a ferment, as threw out a confluence of inflammatory pustules all over him from head to foot."

The physicians opposed it for three reasons: they urged that the inoculated smallpox was not invariably a security against a natural attack of the disease; that many cases occurred in which inoculated smallpox had caused a violent attack ending in death; and thirdly, that inoculated cases, though generally mild, were capable of infecting others, and of propagating the disease far and wide. There is no doubt they were right. The mortality from inoculated smallpox was not as high as in cases of the natural disease, but although inoculation benefited many of those who had this operation performed, it was undoubtedly the case that these inoculated cases propagated smallpox, the multiplication of cases increased the sources of infection, and the average yearly mortality for smallpox rose, amounting in London alone to 1817 at the end of the eighteenth century.

For many years the practice had made little progress, but in the year 1746 the Smallpox Hospital was founded in London for the purpose of inoculating the poor.

It is a curious instance of the slow adoption of new ideas in London that, although smallpox inoculation of out-patients was discontinued in 1808 in favour of vaccination, the inoculation of in-patients was persisted in till 1822, although long before that date vaccination had been generally admitted, not only as rendering the patient immune to smallpox, but as infinitely more safe.

Thus, from the bills of mortality of the latter part of the eighteenth century, when inoculation had become very general, we find that, notwithstanding the preservative effect of inoculation on almost all who had been operated upon, the total number of deaths from smallpox had increased. There is no knowing how disastrous this murderous disease would have become, what great and tragic effects would have been wrought by its ravages, had not the turning-point in the history of smallpox been reached, "a new era in the physical welfare of mankind," brought about by our fellow-countryman, Edward Jenner of immortal fame, the centenary of whose great discovery—vaccination—was celebrated throughout Europe thirteen years ago.

Thomas Jefferson, President of the United States, writing to Jenner in 1806, says: "Future nations will know by history only that the loathsome smallpox has existed, and by you has been extirpated." Let us not be behindhand in testifying to the greatness of Jenner's discovery, and in giving honour where honour is due.

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CONTENTS.

PAGE

***A Lecture on the Radical Cure of Femoral Hernia.** By C. B. LOCKWOOD, F.R.C.S. 321

***Simple Infantile Anæmia.** By EDMUND CAUTLEY, M.D., F.R.C.P. 325

***The Life and Work of Edward Jenner.** By F. M. SANDWITH, M.D., F.R.C.P. ... 328

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A LECTURE

ON

THE RADICAL CURE OF FEMORAL HERNIA.

Delivered at St. Bartholomew's Hospital.

By C. B. LOCKWOOD, F.R.C.S.,
Surgeon to the Hospital.

GENTLEMEN,—On the last occasion I lectured on femoral ruptures and their management.* The indications for the use of trusses was mainly described. Therefore I propose to-day to lecture upon the radical cure. I do this because hereafter some of you will have to perform that operation. To my mind, the operation of radical cure of femoral is not upon the same footing as the radical cure of inguinal hernia. First, as I pointed out to you, the diagnosis of a femoral is very much more difficult than that of an inguinal rupture. Next, the operation for the radical cure of femoral is very much more difficult than the radical cure of inguinal hernia; not only more difficult, but attended with greater danger. I begin with the observations made before the operation, and the reasoning based upon them. The first step is to look at the whole patient. Do not be biassed by what you have read about the incidence of femoral ruptures. I have thrice operated for the radical cure of femoral ruptures in youths. Most of my operations for radical cure in females under twenty-five have been for the cure of inguinal ruptures. Whilst examining the whole patient do not allow your mind to be fixed upon the rupture alone; try to form an estimate of the general condition, the condition of the heart, and the other great organs, especially the kidneys. It is not good surgery to perform an operation for radical cure upon one who has

* *Vide* CLINICAL JOURNAL, July 13th, 1910, vol. xxxvi, p. 219.

chronic nephritis, and yet that has been done. Next, pay attention to the general condition of the abdomen. Those who have attained some age, who have perhaps had a hard life and not particularly good diet, have weakened tissues. In consequence the viscera become lower in the abdomen. The right kidney and colon descend, also other viscera, so that there may be a prolapse of the mesentery, or the transverse meso-colon, of the stomach and pylorus, a condition known as "Glenard's disease," and sometimes called "visceroptosis," or "enteroptosis." Having formed an estimate of the tissues make the patient stand up, and see whether the abdomen sinks in below the chest and bulges out below the umbilicus and at either side of the recti, and whether the hypogastric fossæ lead like funnels towards the inguinal and femoral canals. Those bulgings are characteristic of viceroptosis. When they are seen refrain from operating save in cases of irreducible rupture. And here I would draw your attention to the fact that the term "radical cure" is used ambiguously. At one time it is used to imply that the operation is done with the intention of curing the patient so that the rupture ceases to exist and does not recur. At another time the term "radical cure" is given to the operation although it is merely done to relieve the patient of an irreducible or unmanageable rupture; oftentimes this is all that can be done for femoral ruptures. Having formed a proper estimate of the general condition of the patient, next comes the examination of the femoral region. If the rupture is reducible there is a small soft swelling at the saphenous opening, with an impulse in it. The swelling departs when the patient lies down, but returns when she stands up and coughs. Having descended first of all down the femoral canal, until it is opposite the saphenous opening, and then forwards into Scarpa's triangle, and then upwards over Poupart's ligament, the tumour is felt at the inguinal canal. Thus it is easily mistaken for an inguinal rupture. If you can put your finger into the inguinal canal, then you know that the tumour is outside the inguinal canal. But in females this cannot be done. If the tumour is reducible, then it can be reduced first downwards, then backwards, through the saphenous opening, and finally upwards along the femoral canal. A tumour reduced in that manner will not easily deceive you. Supposing the tumour does not disappear when the patient lies down, cannot be reduced, and is at the femoral opening, then it is still likely to be a femoral rupture. But mistakes are often made, especially in cases of femoral hydroceles, and which may be puzzling to those who are unaware of their peculiarities. A femoral hydrocele is a cyst full of yellow fluid. Its neck is narrow and usually shut off from the abdomen. Sometimes the neck is plugged with a strip of omentum. The cyst itself is the distended sac of a femoral rupture. In an account of a case of femoral hydrocele the author said that very few had been recorded. That is strange, for I have operated upon a number of them. These irreducible femoral herniæ sometimes consist of several cysts, of which most are blind, but one, perhaps, opens by a narrow neck into the abdomen.

At the operation look for the opening into the abdomen, not to gratify idle curiosity, but because the little canal may have a small strip of omentum in it. That omental plug causes fluid to accumulate in the sac. Why does herniated omentum inflame and become adherent? I do not know, but it does. Some would say the inflammation was caused by the pressure of a truss, but I have seen it in those who have never worn a truss. Should the omental strip be overlooked and tied up with the neck of the sac the rupture will not be cured, because more omentum will come down. Besides, an omental band fixed above and below is left, and may lead to internal strangulation. Hereafter you will take pains to search a femoral rupture, femoral hydrocele, or cystic femoral hernia to see whether there is a piece of omentum adherent in the neck of the sac. Sometimes three or four cysts have to be searched to find the opening into the abdomen and to see whether there is anything adherent in it. A fatty femoral rupture is also troublesome from both a diagnostic and from an operative point of view. Femoral ruptures often consist of a minute sac in the midst of a large lump of subperitoneal fat. Also in the neck of the little sac a strip of adherent omentum may be found. A fatty femoral rupture feels like a femoral hydrocele—in truth, femoral hydroceles often have a quantity of fat upon them. Whilst cutting into the sac it is hard to tell whether the fat is upon the outside of the sac or whether it is omentum adherent within it. But the sac is always thickest at its neck, so that it is best to open the neck and see

whether it has omentum in it. Do not trouble about the body of the sac, but set to work upon the neck, where it is thick and firm and can be easily distinguished. Whilst separating the omentum, put pressure-forceps on each piece of the peritoneum as it is cut through, until at last, after an arduous struggle, the omentum is free. But omentum is a vascular structure, and the bleeding may be embarrassing. That bleeding must be absolutely stopped. The omentum must be quite freed, so that it can be pulled down to be transfixed and tied above the bruised and damaged part before being put back into the abdomen. If it be returned and there is reason to believe that it is bleeding, then the abdomen has to be opened and the stump secured. I have done this twice: once whilst operating upon a femoral rupture the omentum slipped back and I thought it was bleeding. I opened the abdomen by an incision above the crural arch and found the omentum was not bleeding. On another occasion, when operating upon a left inguinal hernia, the omentum slipped back and I thought it was bleeding. The abdomen was opened, and the omentum was bleeding and was secured. Fatalities after the radical cure of hernia have oftenest been due to bleeding from the omentum inside the abdomen. A woman upon whom I operated for radical cure of a femoral rupture died. Nothing was ever discovered to account for the patient's death. Enlarged appendices epiploicæ are occasionally found within femoral sacs. The other day an operation was done upon a man who had a femoral epiplocele on the right side, and on the left a fatty hernia with two of the epiploic appendages of the sigmoid flexure adhering within the sac.

Having pointed out some of the difficulties in the diagnosis of femoral ruptures and explained their morbid anatomy, let me tell you something about the operation, which I advise you to learn and perform. It must fulfil three conditions: (1) Complete removal and obliteration of the hernial sac; (2) reduction of the contents of the sac; and (3) repair of the gap through which the sac and its contents descended. Many ways have been devised for fulfilling these conditions. About the removal of the sac and the reduction of its contents there is little dispute, but as regards the closure of the gap in the abdominal wall there are many differences of opinion. Surgeons have endeavoured to close the femoral canal by sewing it up, by putting a piece of muscle and fascia back into it. Roux, by fastening the crural arch down the pectineal ridge by means of a staple driven into the bone. I close it by suturing together Hey's and Cooper's ligaments. Whatever operation you choose it must fulfil the principles of surgery. The incision should be one suitable for exposing the sac. I have seen all sorts of incisions made. Tweedledum will make a vertical incision, Tweedledee a transverse one. I advise you to keep the incision well away from the pudenda; it is then easier to keep the wound aseptic. The proportion of sepsis after radical cure of femoral hernia is higher than after radical cure of inguinal hernia. The sac is superficial, has thin coverings, and is very loose in its bed. It is merely held down by some bands of cribriform fascia, and by one of the external pudic vessels passing over it. So it is easy with the finger to turn it out of its bed. Now you will have before your eyes a structure which may be rather puzzling—a piece of fat, which looks solid, but has a sac in its interior. Whilst cutting into this piece of fat to find the sac you go on and on and wonder when you are ever coming to the sac, and whether you may not injure something in its interior. However, go cautiously on. The neck usually has least fat upon it, and its peritoneum is thickest and easiest to identify. Having opened the neck and found omentum within it, an arduous and anxious separation begins; for these operations are not easy, and not to be undertaken with a light heart. It is not fair to the patient to do a complicated and difficult operation of this kind until you have studied the subject, and it is not quite fair to your own reputation. Having exposed the omentum and got it loose, after infinite trouble, then transfix and tie it. It is a golden rule in surgery never to put a ligature on a pedicle without transfixing it. By following this rule I cannot recall a case of intra-abdominal hæmorrhage. Supposing the contents of the sac are free and safely back, the neck of the sac has to be secured. The neck of the sac is transfixed and tied, like any other pedicle, and then fixed inside the abdomen. The fixing of it inside the abdomen is easy. You leave the ends of the string with which it was transfixed and tied long, to be passed with a curved needle up the femoral canal and pushed through the abdominal

wall, just at the upper edge of the crural arch, pointing the needles rather inwards than outwards. On the whole, it is better to use blunt herniotomy needles. In passing the ligatures the round ligament or the spermatic cord will take care of themselves. The point of the needle, being turned inwards, is not likely to injure the epigastric veins or artery. I cannot recall an instance in which the deep epigastric vessels have been injured. Supposing you have passed the sutures which secure the neck of the sac through the abdominal wall, then you secure the ends. If the suture fixing the neck of the sac be drawn tight it may make a weak spot in the abdominal wall. Therefore draw the ends together, leaving the loop loose, and tie with a jam knot. Sometimes an onlooker says, "You have forgotten to draw it tight." The intention was to leave it loose—not hanging loose, but not constricting the tissues. Having separated the sac, reduced its contents, and secured the neck of the sac, the gap in the abdominal wall has to be repaired. Occasionally one has had to operate upon very large femoral ruptures. When last I lectured I told of one which was nine inches high and eighteen in circumference. The patient was placed in a bed with its foot raised, elastic pressure was put on the rupture, and nearly all of it went back. Afterwards a radical cure was performed. The gap in the abdominal wall was big and easy to manipulate. It was far easier than in the case of small femoral ruptures. So do not be deterred from operating on femoral ruptures because of their size. The femoral aperture is closed by bringing together Hey's and Cooper's ligaments. The latter is not very well known, I am afraid, either to anatomists or to surgeons. The crural arch, of course, is a very familiar object to you all. To the lower end of the crural arch is attached a piece of fascia lata, which begins at the spine of the pubes, runs outwards over the common femoral vessels, and then as the falciform process is continuous with the pubic portion of the fascia lata. The piece of fascia crossing the femoral canal is Hey's ligament.

Cooper's ligament is indicated on this specimen but not properly. It begins at the pubic spine in conjunction with Gimbernat's ligament, runs outwards beneath the femoral canal, common femoral vein and artery, to end upon the ilio-pectineal eminence. The lower edge of Cooper's ligament has fastened to it the pectineus muscle and fascia, to its upper edge the pelvic fascia. Cooper's ligament is so strong that with a hook underneath one can almost lift the pelvis off the table. It has great resisting power. To close the femoral canal pass silk ligatures through the junction of Hey's ligament with the crural arch. When two or three have been passed the ends which emerge from the femoral opening are threaded upon a blunt herniotomy needle, which is passed from inside the pelvis to the outside so as to scrape along the pectineal line and pick up Cooper's ligament from the bone. This is not a very easy thing to do. The finger is placed in the femoral canal, the common femoral vein is pushed outwards with the back of the finger, and with the tip the herniotomy needle is guided along the pectineal ridge, picking up Cooper's ligament. Look at this specimen and see that the inside suture passes through Cooper's ligament, and also through part of Gimbernat's, which runs further out than people suppose. When these sutures have been passed, nothing remains to be done but to bring them firmly together and make as much closure of the femoral opening as you think necessary. The femoral vein will guide the closure of the canal. It must be closed sufficiently to prevent the rupture descending again, but not so as to compress the common femoral vein. The skin suturing completes the operation. The dressing and after-treatment you have abundant opportunities of seeing every day in the wards. I use an outside dressing, like that which was invented by Lord Lister. During the vomiting the abdominal walls act violently, so that air may enter or the dressings be displaced. Next, the outside dressing helps to support the abdominal wall and keep it quiet. Parts kept quiet are less likely to bleed than those which are not well protected. The outside dressing has a layer of jaconet over it. Some patients are careless. On more than one occasion the outside dressing has prevented urine reaching the wound. You cannot take too much care over the preliminary preparation of the patient. Many of the intestinal bacteria are gas-forming. They produce large quantities of gas from the contents of the intestines. The production of gas in the intestines is inevitable. It is certain to take place. You must not assume that I say that all intestinal gas comes from bacteria. But after abdominal operations it is certain that gas will accumulate in the intestines, which will get distended, and the patient may die of obstruction. How shall you prevent that? Prevention is infinitely better than cure. Before the operation clear out the intestinal bacteria and the culture media in which they live.

August 29th, 1910.

SIMPLE INFANTILE ANÆMIA.

A Clinical Lecture delivered at the Medical Graduates' College and Polyclinic.

By EDMUND CAUTLEY, M.D., F.R.C.P.,
Physician to the Belgrave Hospital for Children and to the Metropolitan Hospital.

ALMOST all cases of infantile anæmia are of secondary origin, that is to say, some causative factor is discoverable on careful examination. So constantly is this the case that the occurrence of a primary anæmia in infancy is sometimes denied. The division of anæmia into primary, secondary, and intermediate types is unsatisfactory on clinical and pathological grounds, for multiple causes may be present in particular cases. According to this classification, a primary anæmia is one without obvious cause; a secondary anæmia is one in which a definite cause is discoverable, and is apparently sufficient to account for the degree of anæmia; and an intermediate type is one in which the anæmia is out of proportion to what we are justified in expecting as the normal result of any factor which may happen to be present.

A secondary anæmia is strictly symptomatic. It results from mere loss of blood in hæmorrhage. It is produced by hæmolysis, due to the action of various toxins, or of certain poisons, such as chlorate of potash and lead. Or both these factors may co-exist, as in the hæmolysis and hæmorrhage of hæmorrhagic affections. And in all toxæmias, notably those dependent on gastro-enteric disorders, there is a deficiency in the power of digestion and absorption, and frequently a defect in assimilation and in the supply of iron-containing foods.

But, excluding all these varieties of secondary anæmia, there remains one type, somewhat allied to the chlorosis of later life. For this variety the name *simple infantile anæmia* seems to me appropriate. Its essential basis is a primary deficiency of iron in the fœtal tissues. It is sometimes called *oligosideræmia*, because of the deficiency of iron in the blood.

Symptomatology.—The clinical features of these cases are fairly well defined when the anæmia is marked. In the earliest stages it is probable that the differential diagnosis from a mild secondary anæmia may be impossible. Usually the child is eleven months to three years of age. The youngest patient under my observation for the affection was one of twins, æt. 6 months, who weighed 3 lb. 9 oz. at birth. The child is frequently one of twins or born prematurely. A premature twin is peculiarly susceptible. Generally it is elicited that the child has been brought up by hand, but in a few instances I have seen the affection in the breast-fed. It can occur on a diet of diluted cow's milk, peptonised milk, condensed milk, or proprietary foods, but it has not been my lot to come across it in an infant fed on fresh, undiluted cow's milk, perhaps because so few babies are fed in this manner. Possibly this is partly because the infant is delicate from birth, and the risk of giving cow's milk, even though diluted, has been deemed too great.

The complexion is extremely pallid, with a pale yellowish or even a greenish tint, like that of the chlorotic girl of later life. In severe cases the mucous membranes appear absolutely bloodless. Yet the child is by no means always under weight, though puffy, soft, and flabby. The bowels are somewhat constipated. Digestive troubles and diarrhœa, when present, yield readily to treatment. The spleen is not enlarged. Hæmic murmurs are present.

The mental condition is remarkable for its dulness and apathy. The child does not exhibit the fretfulness and irritability so often present in anæmia of secondary origin.

Blood examination.—The percentage of hæmoglobin falls to 50 or even lower. The number of red cells is little, if at all, reduced. The colour index is low. Poikilocytosis and polychromatophilia are present, and "shadow" corpuscles are often found. But the number of white cells is merely such as is compatible with the youthful age of the child, viz. 10,000–15,000 per c.mm., and the relative proportions of the different white cells are unaltered. Sometimes there is a mild polymorphonuclear leucocytosis.

Pathogenesis.—About twenty-five years ago Bunge estimated the percentage of the various salts in the ash obtained by the incineration of an unsuckled puppy a few hours old and in that of the milk of the puppy's dam. He found that the results were closely comparable. The ash of the milk contained more potassium and less sodium than that of the puppy, probably because during the growth of the animal there is need for a

greater supply of potassium and a less quantity of sodium, for the muscles, rich in potassium, increase in bulk, and the cartilages, rich in sodium, relatively decrease. Put shortly, the maternal milk supply contains the inorganic constituents of food in proportions suitable for the needs of the growing animal.

There was one remarkable exception, namely, six times as much iron in the ash of the puppy as was present in the bitch's milk—0·72 per cent. as compared with 0·12 per cent. of Fe_2O_3. As a rational explanation of this peculiarity, Bunge advanced the hypothesis that the young animal acquires a reserve store of iron, previous to its birth, because of the difficulty experienced by an immature organism in assimilating iron from the food supply. By various analyses he showed that the proportion of iron in the ash per kilo of body-weight was highest at birth, and gradually decreased with the growth and development of the animal. The iron appears to be stored up in the liver, for further analyses showed that the liver of a new-born puppy contained from five to nine times as much iron as that of full-grown dogs. This iron is obtained from the maternal blood *via* the placenta. Possibly the chlorosis of girls is due to the storage of iron in the body in preparation for pregnancy, so that the mother may be enabled to give up iron to the fœtus without suffering ill-effects from the loss.

There is evidence that the iron is stored up by the fœtus during the later months of pregnancy. This explains the fact, which I have already pointed out, that simple infantile anæmia is most common in the premature. Here we find several factors simultaneously in action. The infant has not obtained its preliminary store of iron, and as soon as it has made use of what it has stored up, it begins to develop anæmia. On account of prematurity and general debility it is unable to digest and assimilate its food as well as a normal infant. And its food supply is deficient in iron, even if it is breast-fed, and still more so if it is reared by hand. Bunge's analyses show that there are 3 mgrm. of iron in 100 grm. of dried human milk, and a like amount in cow's milk. It has already been shown that the percentage of iron in human milk is much less than in the new-born animal. But if cow's milk is the diet it is almost invariably diluted, and the supply of iron still further reduced. In addition, the milk mixture is commonly subjected to cooking, to heat of varying degrees, and I think the conclusion is justifiable that the greater the heat applied the greater is the alteration in the organic compounds of iron and the greater the difficulty in their digestion and assimilation. The baby does not receive enough iron to provide the hæmoglobin for the increasing number of erythrocytes. From these considerations it is obvious that the first change in the blood is a deficiency of hæmoglobin.

In normal infants the percentage of hæmoglobin falls during the first six months of life to about 70 per cent. There is an inborn tendency to anæmia. It remains fairly stationary at this level during the next eighteen months of life, and then slowly rises and attains the adult level about the sixth year. Sometimes the percentage of hæmoglobin falls rapidly during the first few weeks of life, remains stationary until about the sixth month, and then slowly rises. This is further proof that the child is unable to obtain from its food supply sufficient iron to supply the requisite amount of hæmoglobin. Applying these observations to the cases in which the anæmia does not develop for some months, it is clear that during the preliminary months the previously stored-up iron is utilised. As soon as this is used up anæmia develops, unless the necessary iron is ingested in a form suitable for assimilation. Hence the greater the prematurity of the infant the earlier will the anæmia develop. Maternal nursing delays its appearance, for human milk contains iron as an assimilable organic compound, unaltered by cooking.

The blood changes.—In order to appreciate the characters of the blood, I must recall to your minds the present hypothesis of the origin of blood-cells. They are all derived from a primitive lymphoid cell of mesoblastic origin. This gives rise to three types of cell. One of these passes through the stages of a large mononuclear cell, a megaloblast, and a normoblast, into the terminal erythrocyte. Eventually the red cells are derived from the red marrow of bones, and any delay or imperfection in their formation, as the result of over-stimulation, for example, leads to the appearance in the blood of immature corpuscles in the shape of poikilocytes, normoblasts, and megaloblasts. The hæmoglobin is less firmly combined

with the protoplasm and shadow corpuscles are frequently present.

A second branch of the primitive mesoblastic blood-cell gives rise to the lymphocytes of the glands, spleen, and thymus. The third cell is a basophilic lymphocyte, very like the large lymphocyte, and from it are derived lymphocytes and myelocytes, the latter of which give rise to the polymorphonuclear cells, eosinophiles and mast-cells (basophiles).

On the assumption that simple infantile anæmia is a primary affection, merely dependent on a deficiency of iron, it is obvious that we shall find no change in the relative proportions of the white cells, unless there is some complication or secondary cause in action. The fact that there is no lymphocytosis beyond that characteristic of the age of the patient is a strong argument against a toxic origin of the anæmia, if we accept the hypothesis that the lymphoid tissues are the chief agents in the destruction of toxins. A toxin would stimulate the activity of the lymphadenoid tissues and produce a certain amount of lymphocytosis. The myeloid and lymphadenoid tissues are differentiated before birth, but as the marrow contains some foci of lymphadenoid tissue, over-stimulation of the marrow might lead to an extra production of lymphocytes as well as of granular cells, in addition to the appearance of poikilocytes, normoblasts, and megaloblasts in the blood. The absence of such changes indicates that there is no undue activity of either myeloid or lymphadenoid tissues.

Diagnosis.—We are now in a position to differentiate simple infantile anæmia from secondary anæmia, one of the commonest affections of early life. If lymphocytosis or polynuclear leucocytosis is present, either the case is not one of simple anæmia or some coincident affection is present. Thus, intestinal toxins give rise to anæmia by causing hæmolysis, and to lymphocytosis by their stimulating action on lymphadenoid tissues. The toxins of congenital syphilis, tuberculosis, malaria, diphtheria, fevers, etc., probably act in a similar manner. In any infantile ailment there is a tendency to lymphocytosis, a reversion of the blood to the more infantile state, because of the excess of lymphadenoid tissue and the ease with which it is stimulated into over-activity.

Stimulation of the bone-marrow leads to increased formation of granular cells (polymorphs and eosinophiles) and a polynuclear leucocytosis, and to the appearance of imperfectly formed cells, myelocytes and hæmatoblasts. Red cells are destroyed. The spleen enlarges in order to dispose of both red and white cells, or in response to the direct action of a toxin.

Hence, the main difference in the blood from that of a secondary anæmia is the absence of a leucocytosis beyond that characteristic of infancy and the absence of splenic enlargement. It is an essential anæmia dependent on incapacity of the blood or blood-forming organs, as opposed to a secondary anæmia due to the action of a factor on the blood-forming organs or on the blood itself, causing hæmolysis. In severe grades of secondary anæmia normoblasts and megaloblasts are found in small numbers. The affection called anæmia pseudo-leukæmica infantum, or von Jaksch's disease, is readily excluded, for it is characterised by splenic hyperplasia and leucopenia, unless there is a leucocytosis from some complication.

Treatment. — It is important to prevent the occurrence of this type of anæmia. Knowing the possibility, and indeed the likelihood, of its occurrence in premature infants, you must be on the watch for its development. The lines of preventive treatment are obvious from the details of pathogenesis. Let the child be breast-fed if practicable. Failing this, see that the diet is suitable to the digestive capacity, and that it contains a sufficiency of iron in the form of organic compounds. It will be of value to remind you of the proportions of iron in various foods and common inorganic preparations. According to a table published in 'The Hospital' (1908), the number of milligrammes of iron in 100 grm. of dried substance is: cow's milk 2·3, potatoes 6·2, green peas 6·8, yolk of egg 18·3, green chicory 22, cabbage 30·5, spinach 40. A little yolk of egg added to the infant's diet in the first year of life is a valuable safeguard against anæmia, and provides other useful organic compounds. Spinach is not always a good means of giving iron, because of the large amount of oxalic acid which it contains.

The best inorganic preparations are the citrate of iron and ammonia, saccharated carbonate of iron, tartrate of iron and reduced iron, in doses suitable to the age of the child. One part of inorganic iron is present, in reduced iron 1, saccharated carbonate of iron 10, tr. ferri perchlor. 28.

Of the various organic compounds advertised one part of iron is present in carniferrin 3·3, triferrin 4·5, ferratin 15·4, iron somatose 50, hæmoglobin 233, hæmogallol 359, and hæmatogen 1420.

I have not found arsenical preparations of much value in these cases. It is best to rely on small doses of a simple inorganic iron preparation and foods such as yolk of egg, the red marrow of bones, meat-juice, potatoes and green vegetables. A caution should be given about meat juice. I have known tapeworm result from its administration; and in hot weather it is very prone to decomposition, even if prepared from fresh meat—a substance not particularly easy to obtain in big towns—and may set up fatal ileo-colitis. Finally, I must impress on you the importance of reducing the quantity of milk in the diet, and replacing it by foods richer in iron. In many cases the reduction of milk and the omission of the proprietary food, which the child is so often given, are essential as a preliminary to the substitution of an appropriate diet. The outlook is extremely good under proper treatment, for the child generally attains a normal colour in three to four months.

August 29th, 1910.

The Use of Vaccines in Septic and Inflammatory Conditions.—Dr. E. W. Cushing, of Boston, considered that vaccines or killed cultures acted only by increasing the resistance of the patient. The opsonic index practically was not of much use in acute conditions. Autogenous vaccines were preferable to stock vaccines, except in certain cases of staphylococcus infection. The best results were obtained in the following order: Infection from colon bacillus, pneumococcus, staphylococcus. The results were not satisfactory with streptococcus in rapid virulent infections. In chronic infections and in cases of little virulence streptococcus vaccines gave satisfactory results, especially in moderate puerperal cases. Vaccines were not successful in acute gonorrhœal infections, but were of much service in gonorrhœal arthritis. Vaccines were useful in the following conditions: Puerperal infections, abortions, infections of bladder and kidneys, appendicitis, abdominal operations, post-operative pneumonia, and abdominal fistula.—*Med. Record*, vol. lxxviii, No. 3.

THE LIFE AND WORK OF EDWARD JENNER.*

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In the long history of medicine certain figures stand out pre-eminently as benefactors of mankind, men whose lives have been devoted to relieving their fellow creatures of at least one of the burdens laid upon them, and to whom we all owe a very special debt of gratitude. Foremost among such men stands Edward Jenner, the man who introduced vaccination, and has saved thousands of lives annually from that most terrible disease, smallpox, in his days the commonest epidemic in all ranks of society. His discovery "was at once the turning-point in the history of smallpox and a new era in the welfare of mankind," and as his practice was adopted, so the terrors of the scourge died before it, till, in this present day, we have almost ceased to realise the blessing that vaccination has brought.

Born in 1749, he lived at a time when the whole of the civilised world was devastated by long years of European warfare, but he had the gratification of knowing, before his death, that he had been the means of saving more millions of lives than had been sacrificed during the whole period of conflict.

Edward Jenner was the younger son of the vicar of Berkeley in Gloucestershire, but he lost his father at the age of five years, after which his education was directed by his elder brother, Stephen. He was sent to school at the age of eight, and there already showed a scientific tendency, for we read in the pages of his faithful biographer, John Baron, that when his schoolfellows were spending their time in play, he was occupied in making collections of fossils and other objects of natural history. From school he was apprenticed to a surgeon at Ludlow, and at the expiration of his term he proceeded to London, in 1770, to enter the house of the famous John Hunter, where he lived for two years to continue his medical studies. John Hunter was at that time surgeon to St. George's Hospital, and was carrying on inquiries into the habits and structure of

* A lecture delivered at Gresham College.

animals. His keen devotion to science, his love of truth, his undying desire for knowledge, and the simple directness of his character, all appealed to the sympathies of Jenner; it was a happy chance which brought these two men together, for their mutual regard developed into a life-long friendship, and Hunter's influence greatly affected Jenner in his subsequent studies. After Jenner's departure from London they carried on a regular correspondence, and although Jenner's letters to Hunter have, unfortunately, been destroyed, we have those written to him by his former master, from which we gather what were their mutual interests and occupations.

Hunter's principle, in his relation to his pupils, was always that of encouragement in research; he never damped the ardour of a pupil, but strove always to awaken in him the love of accurate investigation. The letters from Hunter to Jenner relate chiefly to experiments made on animals to determine certain facts about their habits, temperature and structure, to the study of the laws of migration of birds, and to his geological collection. Jenner sent animals and geological specimens from Gloucestershire to Hunter in London, and there was much writing to and fro about their transport, a somewhat difficult matter in those old coaching days. Whilst Jenner was still with Hunter, Captain Cook returned from his first voyage of discovery, bringing with him a large number of specimens of natural history. Hunter recommended young Jenner to arrange and prepare this valuable collection, which he did with such skill that he was offered the appointment of naturalist in the next expedition, which sailed in 1772. But neither the allurements of travel nor of a London practice shook Jenner in his determination to settle down in the place of his birth, where he had decided his work lay. He had always a passionate love of the country, a sincere affection for the simple country people about him, and no dislike of the laborious work of a country practitioner. But it is to his credit that the country life did not mean, in his case, a life of stagnation. It must have been a matter of extreme difficulty, 150 years ago, to keep in touch with the progress of science in the far-away seclusion of a country village, but to Jenner difficulties were only so many incentives to further exertion, and he undoubtedly kept well abreast with what was going on in the big world. Had he chosen a life other than that of a country doctor, he would, in all probability, not have carried on those observations and studies which led to his great discovery.

There was a popular belief current in many country districts that milkers, who had contracted an eruption on the hands from a disease then common among cows, and which showed itself in pustules on the udder, never afterwards contracted smallpox. Considering how common a disease smallpox was in those days, this was a belief sufficiently remarkable to arrest the attention of so keen an observer as Edward Jenner. It was computed that of every hundred persons, only four reached the age of thirty without having had smallpox, and that one in seven or eight of those attacked died. Besides this, it must be remembered that many of those who survived were left blinded, maimed, or hideously disfigured.

We have an account of how Jenner's attention was first called to the relationship of smallpox and cowpox. During his apprenticeship at Sodbury a young country woman called for medical advice, and the possibility of her having contracted smallpox was mentioned, to which she immediately replied, "I cannot take that disease, for I have had cowpox." This remark, and other rumours he had heard to that effect, rivetted Jenner's attention. He had already had opportunities of witnessing the ravages of smallpox, and, it is said, still had vividly in his mind the miseries attendant on the inoculation for smallpox, which he had recently undergone. Before the inoculation took place there was, he tells us, "bleeding till the blood was thin, purging till the body was wasted to a skeleton, and starving on vegetable diet to keep it so." This treatment was only preparatory, mark you, to the further miseries of the disease itself, which was then inoculated.

From this time onward Jenner constantly brought the question forward and tried to awaken the interest of others in the subject, but he received no encouragement from his professional brethren. He wrote of it to Hunter after his return to Berkeley, and got the characteristic advice, "Don't think, but try; be patient, be accurate."

The country doctors in his neighbourhood had all heard the rumour of this supposed immunity of certain milkers against smallpox, but they considered the evidence inconclusive, and put no value

on it. It required all Jenner's perseverance and courage to continue his researches in the face of constant discouragement and even ridicule, and in the midst of professional work which soon became heavy and all-absorbing as the popularity of the young doctor increased. He was most anxious to get accurate information and to have proofs for his statements before publishing any theories, lest he should arouse false hopes in the minds of men.

But as he collected facts and evidence, and sifted the true from the false reports, his mind became surer. He was riding with a friend one day in 1780 when he propounded his views to him, recounting the history of cowpox, the variety of eruptions affecting the hands of milkers, and which kind of cow-pustule he believed to contain the protective virus. With a deep sense of responsibility he expressed his confident hope that he was on the eve of introducing a means of saving humanity from a terrible scourge, and that, having made his discovery known, he would never rest till the knowledge and practice of vaccination were disseminated all over the world.

"I have entrusted a most important matter to you," he concluded, "which I firmly believe will prove of essential benefit to the human race. I know you, and should not wish what I have stated to be brought into conversation; for should anything untoward turn up in my experiments I should be made, particularly by my medical brethren, the subject of ridicule, for I am the mark they all shoot at."

We have a pleasant picture of him at that time in a description by that same friend, who tells us: "His height was rather under the middle size, his person was robust, but active and well formed. In his dress he was peculiarly neat, and everything about him showed the man intent and serious, and well prepared to meet the duties of his calling. When I first saw him it was on Frampton Green. I was somewhat his junior in years, and had heard so much of Mr. Jenner of Berkeley, that I had no small curiosity to see him. He was dressed in a blue coat and yellow buttons, buckskins, well polished jockey boots, with handsome silver spurs, and he carried a smart whip with a silver handle. His hair, after the fashion of the times, was done up in a club, and he wore a broad brimmed hat."

Jenner was slow in maturing his cowpox studies, and meanwhile we read from his letters how his country life enabled him to continue his botanical observations and studies of bird-life, and to improve his geological collection. He wrote poems of the sentimental vein then prevalent, but happily relieved by the sense of humour which lurks in all his writings.

He founded a local medical society, at which he read papers on various diseases, and where he frequently mentioned his convictions about cowpox, for though he had enjoined secrecy on his friend, from his correspondence we gather he was not reticent on the subject. He was too eager, too anxious to publish his views to be discreet, and he consequently had to suffer from much banter and irritation. The subject became so distasteful to his colleagues that they threatened to turn him out of the meetings if he continued to "harass them with so unprofitable a subject."

In consequence of his papers on natural history he was elected a Fellow of the Royal Society in 1788, and in that year he married a young woman described by his biographer as "elegant in her manners, accomplished in her mind, and possessing an understanding of great vigour." Although she was, unfortunately, often an invalid, it is pleasant to hear of the tender relationship which always existed between husband and wife, and how she comforted and encouraged him in those bad days when, having sacrificed personal fortune and comfort for the sake of his mission in life, he found himself misunderstood and vilified, and often deserted by those whom he had considered his good friends.

In the following year his son Edward was born, who was a source of great anxiety to his parents, for though his nature was gentle and kind, his intelligence was defective. To his father this misfortune was but an added claim to his affection, and his death, at the age of twenty-one, left an indelible scar on the heart of Jenner. Two other children followed, a daughter and a son, who survived him.

By the year 1792 Jenner's practice had become so large that he decided to abandon surgery and confine himself to medicine, and with a view to better his position in that line he obtained the degree of Doctor of Physic from St. Andrews. His gift of human sympathy and his keen insight and powers of reasoning must have made him a

good doctor for those days, though his training, according to modern ideas, was almost *nil*. A year of apprenticeship with a country surgeon, two years of work with Hunter, with whom he presumably visited the wards at St. George's Hospital, and then an entry into general practice. Compare with this our modern scheme of at least five or six years of arduous work at a hospital, with many examinations to pass before a man is qualified to practise. But what Jenner writes to a friend in 1789, describing a successful practitioner, still applies to our days: "A look of significance, a peculiar habit, and a very scanty acquaintance with the human machine, will make a man pass current for a great physician, while, without these auxiliaries, a man with the knowledge of the Hall and College concentrated will be looked upon as a mere pretender."

It is now many years since his attention had first been directed to the relationship of cowpox to smallpox, and although he had published no results, he had spent the time in numerous experiments and investigations. Among other difficulties he found that what was commonly called cowpox was no certain protection against smallpox, but on investigating the matter further he found that cows were subject to a variety of eruptions on their teats, all of which were apt to infect the hands of the milkers, and all of which were popularly called cowpox. It was the true cowpox only that could confer immunity.

The next check he received was to find that milkers who had, undoubtedly, contracted true cowpox were yet, in some instances, later attacked by smallpox. Having ascertained that this was certainly the case his perplexity was great, and many investigators might have abandoned the inquiries as useless. Not so Jenner. By further examination he came to the conclusion that it was only in a particular stage of the pustule that a virus was extracted which imparted the protecting power. He hazarded the guess, since proved to be correct, that vaccinia was smallpox modified by passing through the cow. It has since been proved that "vaccinia is really smallpox which has lost its virulence and its very infectious character, but which has retained the power of giving (when properly used) protection against the unmodified disease."

When he had cleared up these difficulties he proceeded to the next step, which was to ascertain whether the cowpox could be artificially inoculated from one human being to another. On May 14th, 1796, this first attempt at vaccination took place. Matter was taken from the hand of Sarah Nelmes, who had been infected by her master's cows, and was inserted by two cuts into the arms of James Phipps, a healthy boy, æt. 8 years. In due time the vaccine took effect, and the boy went through the slight disease in the proper manner.

But the crucial question now arose, was he proof against smallpox, as Jenner believed him to be? In July this question, so full of anxiety to the operator, was put to the test. Variolous matter, taken direct from a smallpox pustule, was inserted by several incisions. This was not an unjustifiable experiment, because it was then considered the proper course to inoculate smallpox into healthy children to get them through the inevitable disease at a convenient and favourable time.

We can picture the intense anxiety, in spite of his firm faith in the doctrine he was trying to prove, which Jenner must have experienced during the days of waiting. But no signs of the disease followed—the boy was proof against smallpox. "I shall now pursue my experiments," said Jenner, "with redoubled ardour."

His desire to communicate this great discovery to the world at large prompted him to make the experiment known without further delay; he felt almost that everyone who now fell a victim to smallpox did so through his neglect, but again he felt that to arouse so much hope without an absolute certainty was cruel.

"While the vaccine discovery was progressive," he wrote later, "the joy I felt at the prospect before me of being the instrument destined to take away from the world one of its greatest calamities, blended with the fond hope of enjoying independence and domestic peace and happiness, was often so excessive that, in pursuing my favourite subject among the meadows, I have sometimes found myself in a kind of reverie. It is pleasant to me to recollect that these reflections always ended in devout acknowledgment to that Being from whom this and all other mercies flow."

Twenty-two years later his biographer tells the following anecdotes, characteristic of Jenner's care and interest in his patients: "We passed Phipps, his first vaccinated patient. 'Oh, there is poor

Phipps,' he exclaimed, 'he was recently inoculated for smallpox, I believe, for the twentieth time, and all without effect'; and later I found," he says, "lying on his library table a plan of a cottage. 'Oh,' said he, 'that is for poor Phipps; you remember him; he has a miserable place to live in; I am about to give him another. He has been very ill (with consumption), but is now materially better.' This cottage was built and its little garden was laid out and stocked with roses from Jenner's own shrubbery under his personal superintendence."

He wrote a monograph on the cowpox, the original paper, but the manuscript was never printed, and the original is now in the Library of the College of Surgeons.

The first obstacle that he encountered in his further experiments was the disappearance of the cowpox from the local dairies for two years. When it reappeared he repeated his experiments with the utmost care, and in 1798 he published a fuller account of his observations and conclusions in a treatise of about seventy pages, entitled, 'An Inquiry into the Causes and Effects of the Variolæ Vaccinæ, a Disease Discovered in some parts of the Western Counties of England, particularly Gloucestershire, and known by the name of Cowpox.' It has become a classic among medical books. He propounds his theories on smallpox, cites sixteen cases of cowpox which were casually infected, and seven others which were inoculated (these latter including his son Robert, in whose case the vaccination failed), and he comes to the definite conclusion "that the cowpox protects the human constitution from the infection of smallpox."

Before his 'Inquiry' was published, Jenner proceeded to London for the purpose of exhibiting the cowpox, and of proving to his professional colleagues the accuracy of his deductions. For nearly three months he stayed in London with this object, but at the end of that time he returned home bitterly disappointed, having failed to persuade a single individual to receive the vaccine to prove his case. It was natural that there should be some hesitation in accepting so novel a proceeding as vaccination, but considering the fact that Jenner was well known by many men of note in London, and that most people submitted in those days to the dangerous practice of inoculation of smallpox itself, it was surprising that no one should be ready to try this much simpler proceeding, but so it was. Jenner's labours during nearly twenty years had so far brought forth no fruit. But some of the virus Jenner had carried to London was left with Mr. Cline, surgeon to St. Thomas's Hospital, who had a patient with an affection of the hip-joint, and it was thought that perhaps the counter-irritation of the cowpox might give relief. Consequently the virus was injected into the hip of the patient. A few days after he writes to Jenner: "The cowpox experiment has succeeded admirably. The child sickened on the seventh day, and the fever, which was moderate, subsided on the eleventh day. . . . I have since inoculated him with smallpox matter in three places, which were slightly inflamed on the third day, and then subsided. . . I think the substituting of cowpox for the smallpox promises to be one of the greatest improvements that has ever been made in medicine, for it is not only so safe in itself, but also does not endanger others by contagion, in which way the smallpox has done infinite mischief." After this Cline wrote to Jenner for a further supply of vaccine, for the success of this first case had created a great sensation in London, and many learned doctors, who had been deaf to Jenner's pleading, now woke up to take an interest in the subject, though others continued to ridicule the whole proceeding. Mr. Cline, who was now fully convinced of the great value of the discovery, tried to persuade Jenner to come to London and set up in practice there, assuring him that success was bound to follow, with an income of £10,000 a year from the results of vaccination and other practice. But Jenner was not to be moved.

"Shall I," he writes, "who even in the morning of my days sought the lowly and sequestered paths of life, the valley and not the mountain; shall I, now my evening is fast approaching, hold myself up as an object for fortune and for fame?"

But although the great discovery was now launched, public sympathy in England was not immediately forthcoming, and the movement received an early check in consequence of an error committed by Dr. Woodville, the effects of which were difficult to eradicate. In a letter to Lord Egremont, Jenner tells the story thus:

"Dr. Woodville, physician to the smallpox hospital, procured some virus from a cow and inoculated with it several patients at the smallpox

hospital. Fearful that the infection was not advancing properly in some of their arms, he inoculated them with smallpox matter. Both inoculations took effect, and thus in my opinion a foundation was laid for much subsequent error and confusion, for the virus thus generated became the source of future inoculations, not only in the hospital but in London and many parts of the country." (This virus, of course, inoculated into other people, was often not cowpox at all, but was smallpox.) "Hearing a murmur among medical people," Jenner goes on to say, "that the cowpox was not the simple disease I had described, but that in many instances it produced as many eruptions and was attended with as much severity as the smallpox, I went to town with the view of inquiring into the cause of this deviation. Dr. Woodville at once invited me to the smallpox hospital, and very ingenuously told me the whole of his proceedings. The inoculated patients were shown to me, and though some were without eruptions and exhibited the appearance of the true cowpox, others were full of them, and I did not discern any difference between them and the perfect smallpox."

Unfortunately, virus from these patients had been distributed, not only to many parts of this country, but to the Continent, and Woodville had published a book giving the results of his practice, in consequence of which vaccination fell into such bad repute that these trials, Dr. Baron tells us, "had well nigh proved fatal to the infant cause of vaccination."

Dr. Jenner did his best to wipe out the impression made by these mistakes, but it was many years before they were forgotten by a public unable to recognise the greatness of the discovery made by one among them and ready to believe any foolish story told against the practice. He had many opponents, and the discovery was received with derision by some and with suspicion and distrust by almost all. As an answer to objections raised he published in 1799 another pamphlet, "Further Observations on the Variolæ Vaccinæ," in which, in spite of his firm convictions, his genuine humility is shown in the phrase, "Should it appear in the present instance that I have been led into error, fond as I may appear of the offspring of my labours, I had rather see it perish at once than exist and do a public injury."

Besides those who denied the importance of his discovery, there were others who maintained that the fact had always been known that cowpox protected from smallpox, that the knowledge had only been buried in provincial obscurity, and that Jenner's opinions on the matter were of small importance.

In the face of all discouragement he worked incessantly to prove his point, and entered into an enormous correspondence with his many friendly and unfriendly critics. In spite of detractors the practice gained ground, and many people, often ladies, not belonging to the medical profession, instituted vaccination among the poor of their neighbourhood. Thus we read of one Fermor, who carried vaccine from Berkeley to Oxford, where, under the inspection of many learned professors, he vaccinated 326 persons, of whom 173 were subsequently inoculated with smallpox matter and resisted the infection.

Whilst the knowledge was slowly gaining in popularity in England, it was exciting the deepest interest abroad, where it gained a surer footing infinitely sooner than in the country of its birth. In Geneva, Vienna, Hanover, and Prussia it was received with enthusiasm. We read in a letter dated 1799 how Jenner sent vaccine matter to Berlin in compliance with a special request of Queen Louisa of Prussia, the much-loved mother of William I, and great-grandmother of the present Kaiser. In his letter answering this request he states, "Five thousand persons have now been vaccinated, the greater part of whom have been exposed to the infection of the smallpox by inoculation and in every other way, without the least effect."

The city of Bath was the first to form an institute for gratuitous vaccination, and soon after an attempt was made to do the same in London. The Duke of York became patron and requested to have Jenner presented to him, but through mismanagement the institute was short lived, and was succeeded in 1803 by the Royal Jennerian Institution, of which George III became patron.

"Great news from St. James's," writes Jenner; "the King has sent me a very civil message," which refers to the fact that His Majesty had given permission to have the second edition of the 'Inquiry' dedicated to him, and wished to know the author.

The early proceedings of the Royal Jennerian Society were vigorous and prosperous. Thirteen stations were opened in different parts of London, and in eighteen months 12,288 persons were vaccinated, whilst over 19,000 samples of lymph were sent into the country and abroad. The result of these energetic measures was at once manifested in the striking diminution of deaths from smallpox in the bills of mortality. In 1803 they amounted to 1173, the following year they fell to 623. The average annual number of deaths in the fifty previous years had been 2018. The Jennerian Society worked well for a time, but irregularities crept into its proceedings and its influence rapidly declined; in 1808, its finances being exhausted, it came to an end, and the National Vaccine Institution took its place.

Vaccination was making rapid progress throughout the world. In 1799 Jenner's pamphlet reached America, where it was treated with contempt and only slowly became popular. Dr. Waterhouse was the first to teach the new doctrine in the New World. He procured some vaccine from England and vaccinated his own seven children. In order to make the matter as public as possible he took three of these children to the smallpox hospital, where active smallpox matter was inoculated into one of them without infecting him with the disease. This turned the tide of popular feeling, and vaccination was taken up with such zeal by ignorant and unscrupulous people that much harm followed. Matter was taken from ulcers, from smallpox pustules and from degenerated virus, and thus smallpox itself and other diseases were distributed to thousands of people. Dr. Waterhouse, seeing the mischief done, procured a fresh supply of lymph from Jenner, President Jefferson himself set the good example of having his own family vaccinated, and the practice, this time started on a sound footing, gained ground and flourished. Dr. Waterhouse expressed surprise when, a year later, he wrote to Jenner that not a single person had been vaccinated in Philadelphia, where the leading physician had pronounced it to be "too beastly and indelicate for polished society."

The wars of France delayed the introduction of the custom into that country, but by 1800 it had been established, and was carried from there into Spain.

In 1801 a mission of great interest started from England under Drs. Marshall and Walker, to carry the vaccine to Gibraltar and Malta, and from thence to all the countries bordering the Mediterranean. Our fleet was then engaged in the Mediterranean fighting against France, and the preservation of our sailors from smallpox was a matter of supreme importance.

Lord Keith, Vice-Admiral of the Fleet lying at Gibraltar, issued the following order: "Any soldiers, seamen, or marines of the fleet who may not have had the smallpox and wish to avoid that dreadful malady may, by application to Dr. Marshall on board the flagship, be inoculated with the cowpox, which, without pain or illness, or requiring particular diet or state of body, or leaving any marks, effectually excludes all possibility of the patient's ever being affected with the smallpox. By command of the Vice-Admiral." Immediately after the issue of this order, and before many could avail themselves of the opportunity given, the fleet was dispersed, but the enthusiastic doctors, who were giving their services gratuitously, followed the fleet, and we read of their vaccinating the inhabitants of Minorca as well as the seamen of the different vessels. At Malta Dr. Marshall writes: "The smallpox has made its appearance on board the "Alexander" and other ships, several of the seamen had already died of it, and the admiral became alarmed at the probability that so dreadful a calamity should spread through the whole fleet, which, in its present crowded state, would be attended with great hazard to a number of brave seamen, and ultimately the exertions and services of the fleet in the expedition upon which it is now employed." He therefore issued an order for general vaccination. The army, under Sir Ralph Abercromby, when landed at Malta, was exposed to the same danger, and he also gave orders for the general vaccination of the troops.

As their stay in the island was of short duration, Dr. Walker proceeded with the expedition to Egypt, and vaccinated all the seamen and soldiers of the expedition, and "when we come to consider," says Baron, "how many great results have hung upon the issue of this expedition, it is not too much to assert that a practice, which so effectually protected our gallant troops from the dire enemy the smallpox, must have materially contributed to the success of this campaign, at once so glorious and so important to England." The medical

officers of the Navy presented Jenner with a gold medal in recognition of his services to the fleet, for, as stated in the presentation, the seamen had suffered more than other members of the community from the ravages of the smallpox, with a "more than common proportion of deaths."

Jenner was most anxious to extend his practice to India and Ceylon, from whence urgent entreaties for vaccine had reached him, and he succeeded in sending it *viâ* Bagdad. He dipped some lint in vaccine fluid and enclosed it between glasses, which he then dipped at a wax chandler's till they formed a solid ball, which was packed in a box. This packet was safely conveyed to Bagdad, where it arrived with its contents still liquid. Forty people were vaccinated, including the crews of some ships sailing from Bombay. Three weeks later the virus thus reached that port, and twenty or thirty people were vaccinated. In one single instance only did it succeed. From this subject others were vaccinated, and thus it was propagated in the far East, Hindus and Parsees bringing their children to receive the precious preventive.

Jenner, meanwhile, never relaxed his labours in Berkeley and Cheltenham, where he spent some months each year, and where people came from far and near to be vaccinated by him. Of one parish it is told that it refused all offers of gratuitous vaccination till one year, when the people came in great numbers. Jenner inquired the reason of their change of opinion, and was told that the parish found the cost of coffins to be supplied for smallpox paupers to be such a burden that they compelled the parishioners to avail themselves of Jenner's offer. How he must have cherished letters like the following, written by a clergyman: "A few years ago I was in the habit of burying two or three children every evening in the spring and autumn seasons who had died of smallpox, but now this disease has entirely ceased to call a single victim to the grave. Why? I have inoculated (*i. e.* vaccinated) for the cowpox upwards of 3000 persons, and the smallpox is no longer in existence here."

But although his labours increased year by year, his patience with ignorance was inexhaustible, and to his own interests he never gave a thought. Had he kept his discovery secret he might have gained infinitely more credit and made a large fortune by the practice of vaccination alone, but any mercenary thought was utterly foreign to his nature. "The first thing that a stranger would remark," says his biographer, "was the gentleness, the simplicity, the artlessness of his manner. There was a total absence of all ostentation or display, so much so, that in the ordinary intercourse of society he appeared as a person who had no claims to notice. He was perfectly unreserved and free from all guile. He carried his heart and his mind so openly, so undisguisedly, that all might read them." But the demands upon his time and attention were so increased by his labour in the cause of vaccination that he could not devote himself to his practice as a physician, and his private income suffered.

His friends therefore advised him to petition Parliament for a grant in acknowledgment of the benefits he had conferred by his discovery on the nation. A petition was drawn up in 1802, in which he stated that "the series of experiments by which this discovery was developed and completed have not only occupied a considerable portion of your petitioner's life, but have so interrupted him in the ordinary exercise of his profession as materially to abridge his pecuniary advantages"; and he asks "this Honourable House . . . to grant him such remuneration as to their wisdom shall seem meet."

Addington, the Prime Minister, stated that the King recommended the petition, and it was referred to a Committee which was to report: "First, on the utility of the discovery itself; second, the right of the petitioner to claim the discovery; third, the advantage in point of medical practice and pecuniary emolument which he had derived from it." After taking sufficient evidence, the Committee reported strongly in favour of a grant. There was considerable opposition from his detractors, but others pointed out that—"If Dr. Jenner had not chosen openly and honourably to explain to the public all he knew upon the subject, he might have acquired a considerable fortune," and another remarked that, had Jenner kept his discovery a secret, "it was hard to say what sum the House might not be called upon to grant for the purchase of such a discovery." Finally, a grant of £10,000 was voted and passed by three votes only.

The persuasion of some influential friends now broke down the determination he had always expressed of never abandoning his country life, and

in 1804 he took a house in Hertford Street, Mayfair, with the view of starting practice in London. But the attempt proved a failure and disappointment, and a year later he writes: "I have now completely made up my mind respecting London, I have done with it and have again commenced village doctor." The Parliamentary grant was another disappointment. "The Treasury," he says, "still withholds the payment of what was voted me two years ago." Not only was the sum voted delayed in payment, but £1000 was first deducted in payment of official fees!

The Directors of the Vaccine Board took up his cause, which he fought so ill himself, for it was evident that his disinterestedness had seriously crippled him financially, and something must be done to relieve him of his difficulties. A second application was made to Parliament in 1807 for a further grant. The College of Physicians was asked to report on the subject of vaccination, and did it so favourably that the Chancellor of the Exchequer proposed a further sum of £10,000 to be paid to Jenner, this time without any deduction of official fees.

In England smallpox has so lost its terrors that we can scarcely realise what it meant to people one hundred years ago to be delivered from such a scourge. That vaccination came as a deliverer is proved by the tokens of admiration and gratitude which poured in on Jenner from every part of the world. Within ten years of its first introduction it was roughly estimated that upwards of twenty million people were vaccinated, and in consequence the mortality from smallpox was instantly enormously reduced. It is a lamentable fact that England was one of the last among civilised countries to accept the doctrine universally. The opposition was inexplicable and persistent. Hideous prints were circulated to show the result of cowpox inoculation, human beings transformed into cattle, or ejecting cows, a newspaper was published full of ribald falsehoods, and some of Jenner's opponents never ceased to cry down his discovery in the face of all facts. These enemies to vaccination helped to persuade the uneducated that inoculation was still preferable to vaccination, and in London especially the populace generally was bitterly opposed to the new doctrine. Smallpox inoculation of in-patients continued at the smallpox hospital till 1822, in spite of the universal acceptance of vaccination by all doctors of note of every country. When Parliament granted to Jenner the national rewards of the second £10,000, a popular leader sent a messenger to Jenner's lodgings to advise him "immediately to quit London, for there is no knowing what an enraged populace may do."

From its first introduction Jenner had always maintained that one of the advantages of cowpox over smallpox was its non-contagiousness. In inoculation the patient was as capable of infecting others as was any other case of smallpox. He constantly advocated that inoculation should be prohibited by law, and the College of Physicians upheld him when they were asked by Parliament in 1807 to inquire into the causes that hindered the progress of Jenner's vaccination. A Bill was brought before the House of Commons in 1808 to prohibit the use of smallpox inoculation, but after some discussion it was withdrawn. It was not until 1840 that inoculation was made a felony, and the cost of vaccination of the poor was thrown upon the rates.

But this was long after Jenner had passed beyond the turmoil of controversy; during his latter years it was a constant grief and disappointment to him to see how slowly his countrymen accepted the doctrines he was so eager to preach, and which the rest of the civilised world was acclaiming. In 1814 we read of his last visit to London, when the allied sovereigns of Russia and Prussia visited England, and by their special desire the great discoverer of vaccination was presented to them. But he was eager to return to Berkeley, for the death of his wife was imminent.

He never rallied from her loss, never left the country again, and retired as much as possible from active life. In 1818 there was a virulent epidemic of smallpox at Edinburgh, and Jenner was distressed to find that a certain number of vaccinated people became infected. He was forced to the conclusion that vaccination does not in all cases protect entirely from smallpox, though it undoubtedly modifies the disease, and almost always prevents death.

In 1820 he had a stroke of apoplexy, from which he rallied; two years later he had a second stroke, and died on January 26th, 1823. Up to the last he had been actively employed in professional work, in visits of friendship and compassion to his many poor neighbours, in correspondence with his many friends, and in unceasing efforts to further the cause of vaccination.

His purpose was steadfast and unchanging to the last; no discouragement, no failures, could shake his belief in what has since become an accepted scientific fact—the power of vaccination, properly performed, as a sure preventive for some years of smallpox.

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CONTENTS.

PAGE

*A Clinical Lecture on Diseases of the Colon. By W. J. HADLEY, M.D., F.R.C.P., F.R.C.S. 337

*A Clinical Lecture on a Case of Cerebral Tumour. By J. A. ORMEROD, M.D., F.R.C.P. 343

*The Value of Vaccination. By F. M. SANDWITH, M.D., F.R.C.P. 346

* *Specially reported for the Clinical Journal. Revised by the Author.*

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A CLINICAL LECTURE

ON

DISEASES OF THE COLON.

Delivered at the London Hospital.

By W. J. HADLEY, M.D., F.R.C.P., F.R.C.S.,
Physician to the Hospital and to the City of London Hospital for Diseases of the Chest.

GENTLEMEN,—To-day I want to draw your attention to some conditions of the colon. I think it will be best, perhaps, to give you a sort of sketch of what morbid conditions you may get in the colon and then to put special stress on one or two of them. You know that lately there has been a good deal of attention drawn to this organ. Some have thought that possibly the increase of appendicitis is really caused by some colonal disturbance. It does seem as though the number of cases of colon trouble, whether they are mucous, membranous, functional, or ulcerative, has shown a great increase within the last few years. Looked at pathologically or clinically you have practically these conditions to deal with: Catarrhal, hæmorrhagic, ulcerative, membranous, muco-membranous, mucous, and functional disorders. Let us begin with the least important.

Functional.—You must remember in this consideration that the colon is an excretory organ, probably only second in importance to the lungs or kidneys. In many ways one sees this excretory function brought into play. It is used by us in giving certain laxative or purgative drugs. Mercury, *e.g.*, is excreted by the colon, so that you have one of its functional properties coming in to help you in using a laxative drug. Many toxæmic conditions are relieved by the action of the colon in getting rid of poisons, which in their passage cause irritation, with increased action, loose and frequent stools, considerable straining, and possibly tenesmus. As instances one would mention acute

septicæmic diarrhœa and also that occurring in the more chronic toxæmias, gout, and in the last stages of renal or hepatic disease. The colon tries to lessen these conditions by its excretory function. In none of these cases is there any distinct morbid change in the colon.

We come next to *mucous colic.* Writers on this subject prefer to call it mucous colic, thereby signifying that it is a functional disorder. Large strings of mucus are passed, there is often much straining and spasm, there is very rarely any blood, and the colon can sometimes be felt contracted abdominally. As a rule the patients will be neurasthenic women who have generally been constipated and have often abused purgatives. The attacks are intermittent, with sometimes considerable intervals of health in between. When I say health, I mean health as far as the colon is concerned, but they will remain neurasthenic.

Muco-membranous colitis is getting over the border-line from a functional disorder to a pathological state. Here you have a condition allied to the above, but if you examine the strings of mucus that are passed you find fibrinous exudation, showing the inflammatory nature of the condition you are dealing with. The symptoms are, of course, much the same; both rectal and also neurasthenic symptoms are generally present. But in this condition you have a more serious and continuous state of affairs to deal with. The intervals are infrequent, the attacks are more continuous; there is not, in many cases, much free interval at all. The symptoms are preceded very often by some sort of history of constipation or abuse of laxatives, and you will frequently find a large amount of intestinal sand, the grains formed of carbonate of lime. Note the likeness of mucous colic and muco-membranous colitis to asthma, a mucous or a muco-membranous condition. Sometimes asthma is a purely spasmodic affair, at others, an œdema or erythema of the bronchial tubes. You get fibrinous exudation and inflammatory cells in muco-membranous colitis, whilst mucous colic is purely spasmodic.

Then there is a *truly membranous* colitis, which is quite a different thing. This form is seen in diphtheria, pneumonia, and some other specific diseases, or as the effect of certain poisons, like mercury, lead, arsenic, or in some terminal troubles, such as the last stages of renal and hepatic disease. Here you will get a greyish-yellow exudation on the mucous membrane itself, chiefly to be found at the top of the folds, or it may occur in patches over a considerable area on a red, inflamed base. The symptoms are painful diarrhœa, bloody, slimy motions, and tenesmus, and examination of the rectum with your finger or with the rectal speculum, or, in other cases, higher up with the sigmoidoscope, will confirm your diagnosis.

Then there is *catarrhal colitis.* In catarrhal colitis you have a condition which may be an exaggeration of a functional disorder. I mean to say that certain functional disorders can undoubtedly go on to catarrh, so that it may be looked upon as an advanced condition of very severe functional disorder and may stop there. Or it may be the first stage of a much more serious condition—of a true membranous or hæmorrhagic or ulcerative colitis. But as a condition by itself catarrhal colitis is not of any very great moment: it is nothing more than an exaggerated functional condition.

We now come to the *hæmorrhagic and ulcerative form.* I must mention that there are certain specific ulcerations of the intestine which sometimes affect the colon, but these we need not consider in detail. Thus typhoid, syphilis and tubercle beget ulcerations in the colon; they are specific ulcerations about which you may learn elsewhere. Also foreign bodies may produce ulceration in the colon; hard fæces will do the same; cancer or non-malignant growths of the colon can ulcerate. But all these are specific causes of ulceration which we need not consider here, because I want to confine our attention rather more to other groups of ulcerative colitis. Putting aside all these you still have a large group of ulcerative cases about which much has been written lately as to their ætiology, classification, pathology, bacteriology and treatment, and it is to this group that I shall specially draw your attention. It is essential to remember, in thinking over these cases, that however we may attempt to classify the members of this group, some will often pass from one class to another at different stages.

Classified clinically, a case may be at one time catarrhal, at another hæmorrhagic, at another ulcerative in different stages of the same attack. If you classify them bacteriologically the same difficulty arises, for although specific organisms

have been described and claimed in some, yet we must acknowledge that in many long-standing cases there is nearly always a mixed infection, and you do not know the original cause of the trouble. Besides that, you have the difficulty —you might almost say the impossibility—of deciding as to how much of the symptoms or the pathological changes is due to a specific organism, not normal, or common, to the colon, or to some pathogenic power gained by an organism which is normal to the colon. The *coli* group may become very virulent under certain circumstances. Or again, there is the problem of determining how much of the symptoms and pathological changes is due to the interaction of the organisms upon each other. One can imagine that an added organism, although not the cause of the disease, might give a virulence to the *coli* group which would allow changes to take place. It is a more simple matter in an epidemic to investigate the bacteriology, but we are dealing with sporadic cases of disease, which may have many causes, causes which we do not know and cannot put under one heading.

Again, if an attempt be made to classify them anatomically, simply on the basis of morbid anatomy, any condition may be found from a simple catarrhal one up to widespread ulcerative changes extending from the sigmoid to the anus. Any degree of that ulceration may be seen, from a mere pitting or erosion to a denudation of the whole surface, the muscular coat being in some cases invaded, or even perforation resulting.

The next point that I want to bring to your notice is this: some authorities have held that all, or nearly all, these cases of ulcerative colitis are truly dysenteric.

Bearing out this contention we have these facts: (1) Dysentery was formerly very common in England, where, under the name of the bloody flux, it occurred in widespread epidemics with a very heavy mortality. Sporadic cases were also recognised as occurring at times when no epidemic was prevalent. The epidemic is stamped out, but it is quite possible to stamp out an epidemic and yet have sporadic cases.

(2) Epidemics of colitis, having all the characteristics of dysentery, still occasionally break out in institutions, hospitals, and asylums—what has been called "asylum dysentery." Other cases are on record, and many have been carefully investigated; and it is chiefly from that source that bacteriological evidence as to their nature, and as to the possibility of their being of a truly dysenteric character, has been drawn.

(3) The cases that we now call ulcerative colitis, from a clinical standpoint tally almost exactly with sporadic cases of dysentery as described in the seventeenth century. Look up the old authorities and you will get a perfect picture of your modern colitis, in so far as clinical evidence is concerned anyhow. Post-mortem evidences are the same anatomically.

(4) Scrapings of the mucous membrane and sections of the bowel, at any rate in some instances, have shown bacilli indistinguishable from those found in bacillary dysentery.

(5) These bacilli have been further investigated by agglutination tests, and the serum of some cases suffering from ulcerative colitis has agglutinated colonies of true Shiga-Kruse and Flexner bacilli.

Therefore I think we can say, bacteriologically, that what has been called "asylum dysentery," and also many acute sporadic cases, have been practically shown to be identical with bacillary dysentery, whilst the chronic sporadic cases have generally failed to show the characteristic bacilli or the agglutinating tests. But you must remember that in chronic dysentery you have, first, bacillary infection, then an ulcerative process set up thereby, and with that come pyogenic organisms, which possibly take the place of the original bacilli. When, therefore, you come to examine a case, all you find is a pyogenic organism, which is keeping up the trouble, but not the original cause of it.

The importance of determining the bacteriological nature of the disease lies in the question of serum therapy. A serum is now prepared by the Lister Institute, and by the use of a similar serum Shiga claims to have reduced the mortality of bacillary dysentery in Japan from 22 per cent. or 26 per cent. to 9 per cent. or 12 per cent. A reduction of mortality from 22 per cent. to 12 per cent. is an important thing, but distinctly understand that this is in regard to acute bacillary dysentery. I do not think that many of our sporadic cases are caught while they are sufficiently acute to be influenced very much by serum treatment.

Clinically these cases present many varieties.

You may have a simple diarrhœa. Much of our summer diarrhœa may be due to the same organism as bacillary dysentery. It has never been investigated, and I do not see how it can be. But dysentery occurs in the fly time; that is a marked thing, and flies are blamed for colonal infection, sitting (as they may) at one time on fæces, and at another time on food, and so carrying infection from place to place.

Another form is an acute sudden illness with fever, toxæmic symptoms, rapid wasting, bloody, slimy stools up to fifteen to twenty a day, great pain and tenesmus, and a fatal termination in a few weeks from very acute, rapid, ulcerative colitis.

I will quote a case. A woman was admitted, some years ago, hollow-eyed, wasted, cachectic and sallow; she had a dry tongue with aphthæ. She had been ill about ten days with bloody diarrhœa and tenesmus. Fever was only slight (collapsed). Examination showed the rectum ulcerated as far as could be reached, supra-abundant granulations covering the surface, like condylomata. She died in an increasingly toxæmic condition in between three to four weeks from the onset of the symptoms. The colon was found post-mortem ulcerated from the cæcum to the anus. Here there was never any improvement, and the whole course was rapid. There was one continuous downward course, a toxæmic, slightly febrile condition from beginning to end.

In other cases the disease runs a continuous course, but a more chronic one, though there is no intermission of symptoms. It may be very prolonged in some cases, even years in duration. Here is a case. A man, æt. about 30 years, was admitted with bloody diarrhœa of several weeks' duration. There was only slight fever, but considerable wasting. He was very anæmic, the tongue was furred and dry, and the motions up to twelve to fourteen a day. In that case we did an appendicostomy, and irrigation was practised. He improved in general health, took food well, and gained flesh and colour, and for some time he was taking an almost normal diet. But his motions were still bloody and slimy and frequent. The condition of the colon seemed unaltered, and the man died eventually in about five months. Post-mortem the colon was found extensively ulcerated, and in places only polypoid remains of mucous membrane were to be seen. There, as you see, was a very prolonged and continuous ulcerative colitis, never showing improvement from first to last.

In another class of cases you have a chronic intermittent disease. It is intermittent as far as the colon is concerned. Possibly the colon is never cured; I do not know. Here is such a case: A woman, æt. about 35 years, who has had appendicostomy performed, and has had four or five attacks (lasting a varying number of weeks) during the last five to six years. Here is another: A man, æt. about 32 years, who is just now in his third attack; he had the first attack eight years ago, the second six years ago, and the present attack has now lasted three weeks. Sometimes it would seem that there may be intervals of years between the attacks. In the post-mortem room I have seen, in addition to fresh ulceration, scars which showed healing, so that there is no doubt that certain portions of the colon frequently heal in this chronic form.

It is noticeable how frequently cases that get better for a time relapse. It would seem that the ulceration never becomes completely healed, or, at any rate, readily breaks down again. Each attack seems worse than the previous one. That is a common history—first an attack of diarrhœa, then a three weeks' to three months' attack, and then longer and more severe attacks. One also notices that subsequent attacks are frequently determined by some worry, some heavy mental strain, or breakdown in general health. That is perhaps no more than you would expect.

Pain is not by any means always a marked feature in many cases. That is another point I must guard you against. When present it is always most liable to be started by ingestion of food, liquid or solid. When you ask the patient, "What do you take in the way of nourishment?" he will say "I dare not take anything!" "But you must take something to keep you going." "Immediately I put anything into my stomach I get pain followed by a motion." Of course, as you know, the stomach lies on the colon, as it were, or is so close to it that anything put into the stomach is liable to stimulate an already irritated colon, and the patient gets an attack of diarrhœa, which often produces the passage of more or less blood.

The toxic and febrile symptoms vary very much. In acute cases you find that the patients are dis-

tinctly toxæmic. But in other cases you would be surprised to find that the rectal symptoms are a very marked feature, but that there is not very much the matter with the general health. You will sometimes see cases (in literature) in which an appendicostomy or a colostomy has been done and the patient has the colon washed out. He still passes slime and blood, but having an artificial anus he has no more trouble with the colon, and the toxæmia is practically *nil.*

With regard to treatment, you have your specific or bacteriological treatment. I have pointed out that Shiga had claimed a reduction of death-rate in tropical dysentery by the use of his serum, that it has been used in this country, and that the Lister Institute now makes one. The points to bear in mind are these: That the specific organism should be demonstated from a scraping from the mucous membrane (often not found in fæces when it is found in a scraping); that another organism (other than Shiga-Kruse or Flexner) may be found, more or less, in pure culture, and that a vaccine may be prepared from it for treatment and prove more beneficial than the treatment prescribed by Shiga or Flexner. Find out whether the case of colitis is due to the specific organism. It may be a pneumonic, typhoid, or diphtheritic organism. Your serum treatment will depend upon the particular organism you find. Do not blindly use what is supposed to be the specific serum.

Opinions differ as to whether much benefit occurs by its use, at any rate in these sporadic cases, because during the chronic stage the original and causing organism may have gone, and the disease may be kept up by pyogenic organisms. Again, there is the question as to whether the serum should be given *per anum* or *per rectum*, or through an appendicostal opening, or injected under the skin. You can quite understand that the activity of the serum is likely to be markedly altered by passage through the whole of the intestine. When there is an appendicostomy or colostomy, I think the serum is better given through it.

As to the question of symptomatic treatment, the less severe cases will yield to opium and bismuth, preceded by castor-oil and sulphate of magnesia. All you want in those cases is a flow of saline to wash out the intestine.

For more severe cases you certainly need more prolonged treatment. Your second degree of case may be anything from a three weeks' acute toxic condition to many months of prolonged disease. It is always wise to begin with castor-oil and calomel. Calomel is better given in small doses frequently (half to two grains, with a little opium) two or three times a day. It is excreted partly by the colon, and is an intestinal antiseptic. Kino, catechu, and ipecac. have all been used, but without any lasting effect. I have seen some benefit for a time from powdered cuttle fish-bone given in 20 to 30 gr. doses twice a day. Other cases have seemed to be benefited by large doses of tincture of iron.

Your local treatment comprises lavage, which may be done *per rectum*, or from above after appendicostomy or colostomy has been performed. The materials that have been used are very numerous, but I think that modern thought fairly well agrees that what you want is a wash rather than an antiseptic. You cannot expect to anti-septicise the colon. If in the post-mortem room you see the depth of the ulceration, and the depth to which the bacilli and other organisms are found in the mucous membrane of the intestine, you will realise that you are not going to do much good with antiseptics. You may antiseptice the contents of the intestine and so diminish the toxæmia, but you may equally well do it by lavage. You cannot wash out very thoroughly by the rectum, so that if you are washing *per rectum* I think it is wise to use a certain amount of antiseptic—a weak solution. If you are washing through an appendicostomy or colostomy opening you can afford to dispense with antiseptics and use normal saline. Some have used bromide, which, of course, is slightly antiseptic. There are certain other local treatments which you sometimes have to use—nitrate of silver and adrenalin; nitrate of silver when the ulceration is very rife and there is a great amount of blood and slime coming away; and adrenalin when the hæmorrhage is very severe. Nitrate of silver has been used of the strength of a drachm to a pint. I advise half a drachm to a pint.

As to appendicostomy and right-sided colostomy: appendicostomy gives better results than lavage *per rectum*, while colostomy, giving all the advantages of the same thorough means of lavage, also gives complete rest to the colon, as it affords a free opening for the bowel. You have to face

the question, "Am I going to give the patient an artificial anus and do away with the colon altogether?" In view of the severity and pertinacity of the disease, which hardly ever leaves a man when once he is attacked, and of the probability that each succeeding attack will be worse than the previous one, and eventually pull the patient down, I am not quite sure that you had not better do a right colostomy so soon as you have made a diagnosis. Right colostomy certainly gives better results. Indirectly I would mention that the most important points are to diminish the septic condition of the colon, which can be done by frequent lavage, so as to reduce toxæmia to a minimum, and to support the powers of a patient. You must feed him.

There is another reason for colostomy; by its means you wipe out the colon, you have an artificial anus, which will allow you to feed the patient, and you have the upper end of the colon, which will allow you to wash more thoroughly. I do not say you can cure the condition. Many cases have been reported in which all this has been done, and yet cure has not resulted. Moreover, although apparently cured, relapses are the rule.

See to the condition of the patient's mouth (carious teeth, septic gums). Swab the mouth out with a strong solution of carbolic acid; 1 to 40, or even 20, will not hurt if you do it yourself. Do not leave it to anybody else.

The appendicostomy may be left to close of its own accord if the patient recovers. The artificial anus will not close spontaneously, because the diseased colon has a great tendency to contract; it will contract down to the thickness of your finger. Then you are face to face with certain measures. You can try to close the artificial anus, and re-establish the course through the colon, or an ileo-sigmoidostomy can be performed. We need not discuss this matter. There are but few successes recorded.

Functional disorders need hardly any mention, except that I would say a word about the treatment of mucous and muco-membranous colitis in neurotic women. The difficulties of treating these cases are very considerable.

The actual attack in cases of colic may be treated by an enema of warm olive oil, or a laxative or even a purgative may be given. I should not advise the use of cocaine or any sedative. You have to be very careful not to create a drug habit in such women. The application of stupes to the abdomen is often helpful.

In between the attacks the regulation of the patient's life is most important. Insist upon the necessity for some proper employment, forbid the abuse of purgatives, lessen, and eventually do without them altogether. Prohibit their taking all sorts of things; they will often take laxatives of different kinds day after day, and yet you find that they have perpetual diarrhœa the whole time. Then proper diet is most important in these cases. By some it has been thought that all vegetable food should be dropped; others have thought that all meat food should be avoided. A rational, healthy diet is the best means of maintaining what should be a proper state of health. It is much more important to put the diet into the patient in an absolutely non-irritating, finely divided form than to be too careful as to its exact nature. I mean to say that much in the same way as you would feed a case of gastric ulcer you can feed these cases. There must be nothing which will remain as an irritating residue in the bowel.

Undoubtedly in these conditions of mucous and muco-membranous colitis, massage of the colon often does good.

Try to inculcate a proper amount of water-drinking, and thereby you do away with some of the necessity for a laxative. Exercise is most important, and a proper regulation of their lives.

You must remember that you, as medical men, stand between these poor women and a life of absolutely confirmed invalidism on the one hand, or a host of quacks on the other.

September 5th, 1910.

Some Effects of Over-feeding with Fats in Certain Cases of Insanity.—Herman M. Adler says that the organism has provision normally for converting the fatty acid products of digestion into neutral fats. This power may be diminished under certain circumstances. In those cases in which fatty acid is not entirely converted into neutral fats, the toxic effects of the fatty acids are neutralised in other ways. One method by which free fatty acid is neutralised is by conversion into insoluble soap, and when this occurs the body suffers a depletion in its calcium and magnesium content. —*Medical Record*, vol. lxxviii, No. 8.

A CLINICAL LECTURE

ON

A CASE OF CEREBRAL TUMOUR.

Delivered at St. Bartholomew's Hospital.

By J. A. ORMEROD, M.D., F.R.C.P.,
Physician to the Hospital.

GENTLEMEN,—I propose to bring before you to-day a case of cerebral tumour which was under observation in the wards for a very considerable time, and which finally came to post-mortem examination. The principal points about it are these: (1) The nature of the tumour was a sarcoma; (2) its position as found post-mortem was in the right frontal lobe, where it was diagnosed during life. Yet, though it was correctly localised during life, the localising symptoms were very scanty. (3) The patient was operated upon, *i.e.* the skull was trephined, and the dura mater was opened. No tumour was found, but the relief was, as you will see, very complete for the time. However, the symptoms recurred, and finally he died. (4) The first symptoms began in May, 1906. He was under observation on and off in the hospital for the last eleven months of his illness, and the total illness, as estimated from the first symptoms, lasted three and a half years. This is a sort of brief summary of the case. I will now go into the case chronologically, making any remarks in parenthesis as I go along. The first symptoms were fits of a kind, called by the friends "fainting fits." In these fits there were no convulsions, only loss of consciousness. They began in May, 1906, and he used to have about one a week for two or three months. After this he had an interval of nine months without a fit. Then in May, 1906, he had an attack in which he lost consciousness and fell off a ladder and hurt himself. After this he seems to have had an interval of a year without any symptoms at all. In June, 1908, other symptoms began, namely, headache and vomiting. The vomiting, as is usual in such cases, had no relation to food. The headache was intermittent, at any rate at first. It was said to be brought on when the patient went into a bright light. The headache and vomiting became worse, and two weeks before admission he had another fainting fit, which seems to have been the signal for all his symptoms to get worse. On admission the note was briefly as follows: There was nothing in his previous history or in his family history to throw any light on his condition. He had, however, the triad of symptoms which point to cerebral tumour, headache, vomiting, and that other more important symptom, which clinched the diagnosis, optic neuritis. So the existence of a cerebral tumour in this patient was fairly easy to diagnose. But the question still remained, where was it? The localising symptoms were very scanty, being practically limited to two facts: (1) That the optic neuritis was more advanced on the right side than on the left. The value of such a fact as indicating the side of the lesion is still rather doubtful. Some authorities say that optic neuritis begins on the side of the lesion, especially in cases of frontal tumour, but others would not perhaps agree to this. (2) That the man had an incomplete paralysis of the left side of his face. The characters of this facial paralysis were, first of all, that it affected only the lower part of the face, not the orbiculis oculi nor the occipito-frontalis, from which one could infer that it was due, not to disease of the left facial nerve-trunk, but to disease in the opposite hemisphere. Because, as you know, one of the rough and ready ways of diagnosing between facial paralysis due to disease of the nerve-trunk and facial paralysis due to disease of the hemisphere is that in the first case the whole of the face is paralysed, both the upper and lower parts, and in the second case the lower part only. So that in this patient the distribution of the facial paralysis pointed to a lesion of the right side of the brain. Secondly, this facial paralysis had a character which is peculiar and which is not often seen, that is, the paralysis was more marked in reflex muscle movements of his face than in volitional movements. I mean that if you asked him to show his teeth you could notice a certain amount of weakness of the left side, but not very marked, whereas if you made him laugh, the face was definitely and at once drawn up to the right. That is the opposite condition of things to what you find in most cases of facial paralysis. Could any inference be drawn from that? I think one may say that was an additional reason for supposing that the paralysis was not of the nerve-trunk. Further, it is said sometimes (I think the idea was first started by Nothnagel) that paralysis of this kind, more marked in emotional than in voluntary

movement, is one of the symptoms of disease of the optic thalamus. This man had no other symptoms pointing to the optic thalamus, so that I did not feel inclined to think that he had disease of that part. Other symptoms of disease of the optic thalamus might have been involuntary movements of the opposite limbs, incomplete sensory hemiplegia, and spontaneous pains on the other side. But he had none of these things. The peculiar facial paralysis had been noticed about two months before he came into the hospital, he said, and was therefore a comparatively early symptom. And it still remained a solitary symptom, for on admission he had no paralysis of his limbs, no alteration of his reflexes, no affection of ordinary sensation nor of his special senses. Now, though in the absence of symptoms I suspected the tumour might be in the frontal region, I could not say so definitely, and all one could do was to resolve to keep a look-out for any fresh symptoms which might arise. We were certain about the existence of a tumour, though we could say little more about its position than that it was probably on the right side of the brain. There was nothing to throw any light upon the important question of the nature of the tumour. Now medical treatment in such cases resolves itself mainly into the administration of anti-syphilitics, which can do no harm if a tumour is not syphilitic, and, if it be syphilitic, may effect a cure. So we put the patient upon iodide of potassium, half drachm doses, three times a day, partly in the hope that the disease might be syphilitic, and partly because he had severe headaches, and iodide of potassium will sometimes do good to the headache of cerebral tumour, quite apart from syphilis. We tried other things also to relieve the headache, such as phenacetin, caffein, and drugs of that class. And for a time these measures seemed to do good, but their effect was only temporary. We kept a watch for the development of further symptoms, and the next symptom which occurred was a rather puzzling one. About the end of January or the beginning of February he developed a diplopia. This diplopia was found, on examination, to be due to weakness of the right external rectus muscle, indicating some interference with the function of the right sixth nerve-trunk. You might think that that would be sufficient ground for placing the disease somewhere in the neighbourhood of the pons or base of the brain. But I must remark that, tempting as that sounds, it is not always a correct inference, because it seems that for some reason or another the sixth nerve-trunk is particularly liable to paralysis in cases of cerebral tumour, even though the disease does not involve the parts through which it runs. And as a matter of fact the diplopia disappeared again, showing that the sixth nerve had only been affected in some indirect way. The headache continued and became worse, and the patient became rather dull and stupid. So far I have not said anything about the patient's mental condition. You might expect that in frontal tumour the mind would be affected. But up to this point there had been very little to say in that respect about our patient. He seemed to be fairly sensible, he told us about his illness, and he did not exhibit any mental irregularity that we could fix upon. I do not think his friends were of opinion that his mind was affected at all. However, at this stage he began to get rather dull. That of course might have been due to his headaches and to the general spread of the tumour rather than to its actual position. The next symptom which we particularly noticed was a certain fine tremor of his hands. This is a symptom of frontal tumour which has been emphasised by my colleague Dr. Grainger Stewart, in a very excellent paper on the subject of frontal tumours which he published in the 'Lancet' for November 3rd, 1906. He says that in frontal tumour you may observe a fine tremor of the hands, particularly in the hand on the same side as that on which the tumour is. We looked out for this sign day after day in our patient, but it did not appear. But finally, about the middle of March we did notice that he had a fine tremor of the right hand, and a less distinct tremor of the left hand when he held them out. The next symptom, which we noticed for the first time a few days later than the tremor, was that the abdominal reflex on the left side had disappeared. I say "disappeared" because we had examined him repeatedly to ascertain what the state of that reflex was, and had always found it normal. Absence of abdominal reflex on one side may be found in cases of hemiplegia where the motor tract to one side of the body is affected. In this case, assuming the tumour to be in the right frontal lobe, the loss of abdominal reflex on the left side was taken to indicate backward pressure upon the right internal capsule; and we

shall see later on that as the case progressed actual weakness of the left side developed. His headache became persistently worse and the symptoms appeared to increase steadily, and as the anti-syphilitic treatment by means of iodide and mercury seemed to bring about no permanent benefit, I thought the time had arrived to ask Mr. Bowlby to see him. And Mr. Bowlby agreed with me that it would be best to trephine over the right frontal region. That was done on April 2nd. Mr. Bowlby made the trephine hole which you see here in the skull. He found that the skull there was very thin indeed, almost of the thinness of paper, but since the operation you see a good deal of new bone has formed round the hole. The dura mater was laid open, and then the brain bulged, clearly indicating that it was under pressure. But no abnormality of structure could be seen. On looking further down Mr. Bowlby thought the brain was not pulsating as it should do. So he made a further opening and slit up the dura mater further. Still no tumour was seen. You may think it was very disappointing to see nothing. But as a matter of fact I did not expect to see much, for I strongly suspected that the tumour lay, not in the cortex, but in the brain substance beneath. Anyhow, no tumour was seen, and therefore nothing was removed, and the operation resolved itself into an operation for the relief of pressure—"decompression" as it is called. Yet the result was very striking. In a very short time the patient expressed himself as feeling perfectly well. He lost his headache and his vomiting. He lost, too, the tremor of his hands, and the abdominal reflex came back again. He did not, it is true, lose his optic neuritis, but as that neuritis was not such as to interfere with his sight the presence of it did not trouble him. The operation having been done on April 2nd, he was able to go home well, as he thought, on April 20th. One could hardly anticipate that he would be permanently well. He came into Matthew Ward again on August 6th and stayed there for a short time, namely, until September 2nd —not because he felt seriously ill, but because the headaches had recurred more or less, and he wanted to see whether anything more could be done for him. His headaches continued, though slight in degree, until August 27th; at that date he began again to have violent headache and vomiting. Whether it was that he was disappointed with the recurrence of these symptoms I do not know, but he wanted to go out, and did leave the hospital. It was unfortunate, because he soon became still worse. Dr. Beauchamp Hall, of Islington, wrote me an interesting account of the patient while he was at home. On September 26th (he says) the chief symptom was severe paroxysmal headache; one attack, at least, of these headaches was experienced each day, lasting anything from four to twelve hours, beginning generally over the left occiput and then becoming general, followed by severe ocular pain and pain in the superior maxilla on both sides, with prostration. The attacks of pain were accompanied by sickness. His blood-pressure under the administration of a grain of erythrol a day had sunk to 115 mm. of mercury. The headaches were always accompanied by some throbbing of the carotids and increased tension in the hernia cerebri which now had formed. There was more or less marked œdema of the right infra-orbital region, though without proptosis. There were no ocular symptoms except the optic neuritis, but there was tinnitus in the left ear. A new symptom had appeared—paresis of the left side and increased deep reflex on the left side, with some little ankle clonus and sometimes extensor plantar reflex on the left side (so that by this time he showed very definite evidence of pressure in the pyramidal tract). He was now admitted for the third time. The condition when he came in was very much that which has just been described. There was weakness of the left side, not very marked, but gradually increasing; the plantar reflex on the left side was sometimes extensor, sometimes not. The mental condition had become peculiar. The memory was not impaired either for recent or remote events, but he was rather irritable and emotional and lacking in self-restraint. There were no hallucinations or delusions. Sometimes he did not seem to know whether he was in hospital or not. He was slow in answering questions. Mr. Bowlby saw him with me once or twice, but his general state did not seem to be such as to make a further operation justifiable. Therefore no further operation was carried out. Towards the end of October his temperature rose and his mental condition seemed to get worse. He was at this date passing his urine and his motions in the bed, and he seemed generally much more stupid than he had previously been. These symptoms, however, passed

off again, his mental condition became rather better, and his temperature became normal. Nevertheless he died on November 15th. A post-mortem examination was made, and this is the result: You see there is a tumour nearly as large as an orange in the right frontal region. It has come to the surface (probably as the result of pressure from within) at the trephine hole. I have already told you that it did not reach the surface at the time of operation. Microscopically it is a sarcoma, and although it looks in parts as if it had a certain limiting layer, yet I think it is very doubtful whether it could at any time have been removed without doing a great deal of damage to the brain-substance. The tumour bulges into the anterior cornu of the lateral ventricle and has pushed backwards this anterior cornu, and also the caudate nucleus and the internal capsule. To this pressure no doubt was due the left hemiplegia, which developed late in the course of the case. The optic thalamus is not diseased. So that the diagnosis of tumour involving the right frontal lobe was correct. The surgical operation also was justified by the relief which it gave to the distressing headache. Quite as much good, I think, was done by the simple operation for decompression as would have been done by an attempt to remove the tumour.

September 5th, 1910.

THE VALUE OF VACCINATION.*

By F. M. SANDWITH, M.D., F.R.C.P.,
Gresham Professor of Physic.

IMMUNITY from smallpox can be acquired in three ways: (1) A previous attack of the disease usually, but not always, protects an individual from a second attack; (2) inoculation, now forbidden by law, secures protection; and (3) vaccination, or inoculation of smallpox from the calf, if repeated sufficiently often, protects completely from smallpox.

Vaccinia, or cowpox, is an infectious eruptive disease of cows affecting the udder and teats, and is spread among cows by the hands of the milkers.

After inoculation of a human being with vaccine lymph there are no symptoms for three days, during which the disease is incubating. Raised red flat papules are then seen at the place of inoculation and on the fifth day become vesicles, which reach their maturity on the eighth day. The spots then become cloudy, yellowish and purulent, while the skin surrounding them is red and swollen until the tenth day, when the spots begin to dry up and form a scab, which eventually falls off, leaving well-marked pitting behind it.

The general symptoms which accompany these local signs are slight fever, headache, loss of appetite and sleeplessness, while some children also show a red rash.

You must observe the resemblance of the stages of development of smallpox and cowpox, and you are aware that an attack of the one disease protects for some years an individual against the other disease.

It is not easy in this country to find a large number of people who have had the smallpox, but in Egypt I have had the opportunity of vaccinating many negroes and Nubians, who told me that they had been inoculated with smallpox as children. They willingly submitted to vaccination but assured me that it would not take, and they were right. They developed no sore arms, though Egyptians and Europeans, vaccinated on the same day with lymph from the same calf, showed the usual effects of successful re-vaccination.

The identity of the two diseases, smallpox and cowpox, has been difficult to prove, but Dr. Copeman, an inspector of the Local Government Board, has lately been able to show that vaccinia is nothing more nor less than smallpox modified by transmission through the calf.

As it is illegal to inoculate man with smallpox he inoculated some monkeys, and from the vesicles thus produced he secured smallpox "lymph which after passage through several calves ultimately produced typical vaccine vesicles, from which, again, children were successfully and typically vaccinated.

"The importance of the results obtained from this experimental work consists in the fact that they afford scientific confirmation to an extent previously unattained of the definite relationship of vaccinia to smallpox." (Copeman.)

Let me very briefly mention some of the facts which prove the value of vaccination.

(1) The marked fall in mortality from smallpox in all countries dating from the introduction of vaccination.

Since 1800 the mortality showed a considerable decline, until there was a slight rise about twenty

* The fourth of a series of lectures on smallpox and vaccination, delivered at Gresham College.

years later, when there was a large number of persons requiring re-vaccination.

(2) The vaccinated suffer from smallpox much less than the unvaccinated, as shown by two recent instances: At Warrington only 4·4 per cent. of the vaccinated children exposed to infection were attacked, compared with 54·5 per cent. of the unvaccinated; while at Dewsbury 10 per cent. of the vaccinated and 50 per cent. of the unvaccinated suffered.

(3) The age-incidence of smallpox is quite different since the introduction of vaccination. In olden days nearly all the children took it, but to-day we find in towns where infantile vaccination is thoroughly carried out that most of the smallpox patients are adults. On the other hand, in Gloucester, where 85 per cent. of the children had never been vaccinated, 64 per cent. of the sick in the last epidemic of 1898 were children under ten years of age.

Dr. Ker contrasts these figures with well-vaccinated Glasgow in 1900, where out of 1730 patients only 5·5 per cent. were under the age of ten, and of these victims two thirds had never been vaccinated.

(4) People who have been once vaccinated, but are not protected by re-vaccination, have a much lower death-rate, when attacked by smallpox than those who are unvaccinated. For instance, in the London epidemic of 1900–1, 9659 cases were treated in the smallpox hospitals of the Metropolitan Asylums Board, and of these 11·5 per cent. of the vaccinated died, while 33·1 per cent. of the unvaccinated succumbed.

These figures show you that vaccination does not always protect from smallpox; it is important that the vaccination should be efficient. Efficiency depends on the character of the marks and the number of the marks. How often has an anxious and affectionate mother said to me, "I hope you will vaccinate my baby in only one place." But when I have explained to her the statistical reason for making four or five sore places on the baby's arm or leg, she has always said, "Please do it as you think best for my baby." Let me take, again, figures from more than 11,000 cases treated in the Metropolitan Asylums Board hospitals; the death-rate per cent. of those with only one mark was 13·5, with two marks 8·4, with three marks 6·4, and with four or more marks 3·8.

(5) Systematic re-vaccination, which was not dreamed of in Jenner's time, protects any individual from smallpox.

In Germany, where re-vaccination for some years has been systematic and efficient, smallpox is almost unknown; the few individuals who suffer from it are foreigners who have not been re-vaccinated. It is therefore not necessary to have smallpox hospitals, and Berlin has only twelve beds for smallpox, while London is obliged to keep up an establishment of 2040 beds.

The German law requires vaccination in infancy, re-vaccination during school life, and the men are again re-vaccinated when they enter the Army.

The most striking instance of the protective power of re-vaccination is to be found among the nurses in smallpox hospitals.

The Epidemiological Society, while investigating this point, found that of 734 attendants in the hospitals of the Metropolitan Asylums Board, 79 had already had smallpox, and 645 had been successfully re-vaccinated before they entered upon their duties. Not one of these 724 people, in daily contact with the sick, took smallpox. There remain ten who had not been re-vaccinated, and every one of them caught smallpox.

No one is in such intimate contact with fever patients as the nurses who have to wash the sick, dress their sores, and carry away their soiled bedding. Nurses often catch from their patients scarlet fever, diphtheria, measles, typhus, and typhoid fever. What protects nurses from smallpox? Only one thing, re-vaccination. I could give you several similar instances, but I will only mention one more. At the Homerton Hospital, where I first learnt something about smallpox, during the years 1871–77, 366 attendants were employed, and only one took smallpox. She was the only individual who had not been re-vaccinated.

On the other hand, at Leicester, in 1892–3, where disbelief in vaccination and re-vaccination was rampant, six nurses out of forty refused to be re-vaccinated: one of these six caught smallpox and died of it, while four others contracted the disease and recovered. The sixth disbeliever, who escaped infection, was the matron, whose relations with the patients were of a less intimate character than those of the nurses.

I believe that any conscientious objector could

at once be converted to belief in vaccination if he could see, as I have done, healthy children living for weeks in a smallpox hospital, and obliged to be there because there was no one else to look after them. One thing, and one thing only, protected these children—they were vaccinated directly their mothers were found to have smallpox.

A few weeks ago I received from Prof. Osler a pamphlet published by the Pennsylvania Department of Health, which gives incontestable evidence in favour of the absolute immunity furnished by vaccination. The following extracts from the official report of Major R. S. Woodson, Surgeon to the United States Army, tell the story:

"On October 20th, 1898, I was ordered by General Wood to proceed to Gibara, Cuba, to examine into the sanitary condition of the district and select a camp site for military occupancy. Up to this time no American soldiers had entered this district, and no information had been received of the frightful amount of smallpox prevailing there. The disease had assumed epidemic proportions in March, 1898, and had steadily progressed until, upon our arrival, there were, upon conservative estimate, 4000 cases with 30 deaths a day in the district—200 miles long by 75 miles wide. A sad incident gives an idea of its virulence and magnitude. Upon my first visit of inspection to the small town Aguas Claras I was shown a house with seven victims in it. Upon my return in two or three days to convey them to the nearest isolation hospital I found them all dead and the town completely deserted, with its neighbouring cornfields converted into graveyards.

"The cause of the virulence and magnitude of the epidemic was due to the fact that for thirty years there had been almost continual warfare and political unrest, and consequently but very little vaccination had been done, thus allowing a large unprotected population to grow up, offering a fertile field for disease invasion.

"Our methods of control were those usually adopted for the suppression of epidemics, but our principal reliance was placed in vaccinations, of which we recorded 30,000. As a result we obtained phenomenal success in controlling and checking the epidemic, until, in January, 1899 (just two months), we destroyed by fire the large Spanish hospital in Holguin City, in which we had treated 501 cases, to be followed shortly by the similar destruction of the now needless isolation hospitals at other centres of infection.

"As stated above, careful investigation of the mortality statistics of the district for the preceding six months showed, during a great portion of the time, thirty deaths a day from smallpox. We conservatively estimated the epidemic at 4000—certainly the greatest epidemic of modern times.

"Dr. Garcia's statistics show that, before we came, only 5·66 per cent. of total cases had been vaccinated, and of these the mortality was *nil*, and the cases all discrete.

"To illustrate further the efficiency of vaccination and to prove the fact of a virgin population, we obtained 95 per cent. of successful vaccinations with 20,000 ivory points that General Wood obtained in person from the United States, causing a lowering of mortality-rate to 9·6.

"The effect of vaccination seemed to be to check instantly the epidemic as though it had been swept from the district. The crowning feature of all in proving the efficiency of vaccination was the fact that the entire regiment, 2nd U.S. Volunteers (Hood), all protected by vaccination, encamped practically in the town of Holguin and Gibara, mingling freely with the inhabitants, exhibited an absolute immunity against smallpox, as not a single case occurred in this regiment during the six months' service in the district.

"Upon reporting the frightful sanitary conditions of the district, and upon receipt of orders placing me in charge of the epidemic, General Wood stated to me that he would withdraw the regiment until the district could be put in good sanitary condition if I recommended it. So firm was my belief, however, in vaccination that I recommended that the regiment be allowed to remain on duty in Holguin. It was a bold procedure, but the subsequent results supported it, and in my opinion furnished one of the strongest proofs of the efficacy of careful and efficient vaccination."

I need add nothing to this clear account, except to say that General Wood, once a medical man himself, considers that the total number of cases stated by Major Woodson can be safely doubled. It appears that the regiment of volunteers was specially re-vaccinated before it was sent into the infected district.

I think I have proved to you that it is nonsense

to say that vaccination and systematic revaccination do not protect against smallpox. That is the first of the objections raised by anti-vaccinationists. Next it is urged that vaccination causes erysipelas and other skin diseases; that may be quite true if the operation is not done in a cleanly way and if the necessary wounds are not kept free from dirt. Some doctors have been, and are, careless about impressing this latter necessity upon those in charge of the child.

Again, I have heard it urged that the bad teeth of the present day are due to vaccination. Teeth are undoubtedly bad, but they are no more caused by vaccination than they are by electric lighting or the extension of underground railways. A dreadful human disease, syphilis, has occasionally been conveyed by lymph to a healthy child, and that is why Jenner's method of arm-to-arm vaccination has been discarded since 1899 for the safer calf lymph which is now invariably used by public vaccinators, and I hope by all private vaccinators.

Before the calf lymph is issued care is taken to be certain that the calf which furnished the lymph was not suffering from tubercle or any other communicable disease, and the lymph is mixed with glycerine, which destroys all extraneous germs. Lastly, the objection is often raised that by making vaccination compulsory we are interfering with the liberty of the subject. This is a strange stumbling-block in a country which has cheerfully submitted for some forty years to compulsory education of children. It has been pointed out that the alternative to the practice of vaccination must be sooner or later an outbreak of smallpox, which for the safety of the community can only be stemmed by compulsory isolation of the sick and a prolonged quarantine. This obviously entails a much greater interference with the liberty of the subject than simple vaccination.

In some respects we have had to modify the belief that Jenner had in vaccination. We now know as a fact that vaccination confers immunity from smallpox for a certain number of years only; re-vaccination is necessary for perfect and permanent safety. In epidemics it is proved that vaccination in infancy protects a person for five years, makes him almost always immune for another five years, and that after ten years the proportion attacked increases with each year, rendering the subject more liable to attack, although even the one vaccination in infancy prevents the attack, with the exception of a very few cases, becoming fatal. This is a very important lesson which we have learned in recent epidemics and from the example of other countries where the vaccination laws are more stringent than ours.

In Germany smallpox is now almost extinct. Not only is each child compulsorily vaccinated in infancy, but it is re-vaccinated in its twelfth year, and this has been enforced since 1875. Since that year smallpox as an epidemic has ceased to exist. There are a few deaths annually, at the rate of one per million on an average, but these deaths occur almost exclusively on the Russian and Austrian frontiers. Smallpox was as prevalent in Germany in pre-vaccination days as anywhere else in Europe (King Frederick William III of Prussia, in a despatch of October, 1803, states that 40,000 people died annually of smallpox in his kingdom), and even in 1872, when vaccination was not systematically carried out, 2·7 per thousand of the population died of it. But this led to sterner measures, with the excellent result I have quoted.

In Jenner's day vaccination was carried on from arm to arm, but this led, in some instances, to the propagation of other diseases. It was originally thought that lymph from an unhealthy subject could convey no other poison but the vaccine poison, but this supposition is now proved to be wrong and has been abandoned.

The lymph supplied by vaccine institutes "is cultivated by the vaccination of a succession of calves. The buildings in which such calves are stalled are built with every possible care. The calves selected are carefully examined, tested to prove that they are free from tuberculosis, and then watched for some days to make sure that they are in good health. When vaccinated the routine followed is the same as that adopted in surgical operations. After the lymph has been taken the calves are slaughtered, and the carcases minutely examined by a veterinary surgeon specially appointed for the purpose, to make certain that no disease is present. If any abnormality is discovered in a calf the whole of the lymph taken from that animal is destroyed. Lymph which is selected for use is prepared with the most minute attention to details, and is treated with glycerine for some weeks to destroy all

foreign germs, leaving the lymph free from other organisms, but still retaining the active principle of cowpox."

Besides these necessary precautions it is, of course, essential to keep the wound clean. Among dirty people this is not always the case, with the result that the arm becomes dangerously inflamed, and there is a general temporary poisoning of the whole system. With properly prepared animal lymph and attention to cleanliness of the wound, vaccination is a safe, as well as a necessary, operation.

"The dangers of vaccination have been enormously exaggerated by the opponents of this measure. In the Philippine Islands within the past few years there have been performed by the United States sanitary authorities 3,515,000 vaccinations without a single death or any serious post-vaccinal infection. When we consider the thousands on thousands of vaccinations performed, even on the unclean and under unfavourable circumstances, and note how rare it is for any serious complication to develop, we are justified in concluding that the risk attending vaccination . . . is practically a negligible quantity."

The conscientious objector and the anti-vaccinator brought so many and such various charges against the practice of vaccination that a Royal Commission was appointed in 1889 to inquire into the efficacy of vaccination as a protection against smallpox, and to search into the alleged danger of the operation. These Commissioners included such well-honoured names as the Right Hon. Lord Herschell (Lord Chancellor and President of the Commission), Sir James Paget (Sergeant-Surgeon to Queen Victoria), Joseph Bright, M.P., Prof. Michael Foster, and others. Their inquiries were exhaustive and lasted for seven years, and the conclusions they finally came to were these:

"It has appeared to us impossible to resist the conclusion that vaccination has a protective effect in relation to smallpox.

"We think:

"(1) That it diminishes the liability to be attacked by the disease.

"(2) That it modifies the character of the disease and renders it (*a*) less fatal, and (*b*) of a milder or less severe type.

"(3) That the protection it affords against attacks of the disease is greatest during the years immediately succeeding the operation of vaccination. It is impossible to fix with precision the length of this period of highest protection. Though not in all cases the same, if a period is to be fixed it might, we think, fairly be said to cover in general a period of nine or ten years.

"(4) That after the lapse of the period of highest protective potency, the efficacy of vaccination to protect against attack rapidly diminishes, but that it is still considerable in the next quinquennium, and possibly never altogether ceases.

"(5) That its power to modify the character of the disease is also greatest in the period in which its power to protect from attack is greatest, but that its power thus to modify the disease does not diminish as rapidly as its protective influence against attacks, and its efficacy during the later periods of life to modify the disease is still very considerable.

"(6) That re-vaccination restores the protection which lapse of time has diminished, but the evidence shows that this protection again diminishes, and that, to ensure the highest degree of protection which vaccination can give, the operation should be at intervals repeated.

"(7) *That the beneficial effects of vaccination are most experienced by those in whose case it has been most thorough.* We think it may fairly be concluded that where the vaccine matter is inserted in three or four places it is more effectual than when introduced into one or two places only, and that if the vaccination marks are of an area of half a square inch they indicate a better state of protection than if their area be at all considerably below this."

In the lantern-slides which I will presently show you you will see the unfortunate results of the anti-vaccination campaign, which continues unabated.

The conscientious objector, by Act of Parliament of 1898, is allowed exemption from vaccinating his children if he conscientiously believes that vaccination would be prejudicial to their health. Since 1907 this has been made even easier for him; he no longer has to appear before a magistrate to declare his conviction, and since this change in the Act the growth of conscientious objection has been extremely rapid. In the year 1908 alone there were 162,800 such exempted

infants. In some parts of the country the unvaccinated considerably exceed the number of vaccinated, and it is terrible to think what will happen should smallpox once get a footing now in this country. A suitable soil for a great and terrible epidemic is now prepared.

As Dr. Leonard Cane says: "The attitude of the Local Government Board is inconsistent. Compulsory vaccination is either a protective measure beneficial to the whole community, or an act of doubtful utility imposed against the will of a large section of the people, to whom it appears but an unwarranted intrusion of an officious State into the freedom of the home. If the latter, then these laws are unjust, and the sooner they are repealed the better it must be for all; but if the Government really have faith in the experience of practically the entire medical profession, both in this country and abroad, and the conclusions arrived at by its own Royal Commission after nine years' continuous investigation, then it is its duty to safeguard the national health by increasing the stringency of the vaccination laws, so that in this country, as elsewhere, the disease, which until a century ago was such a terrible scourge, may be entirely stamped out, and the risk of such an expensive outreak reduced to a minimum."

Let us briefly look at the history of some recent epidemics of smallpox in England. During 1901 and 1902 there were 9223 cases of smallpox treated in the hospitals of the Metropolitan Asylums Board of London. Among the vaccinated the deaths were 10·1 per 100 cases, among the unvaccinated the deaths were 33·5 per 100. In 1896 there was a severe epidemic of smallpox at Middlesborough, with a total of 1411 cases, in 1898 another in Gloucester with 1979 cases. It is well to look at the effect on the children first, because in their case, surely, we are specially responsible when they have been sacrificed to prejudice. Were the vaccinated attacked as well as the unvaccinated? Yes, those who had been vaccinated in infancy only were, to a limited extent, attacked. In Middlesborough there were forty-three such cases, but not one death, and in Gloucester there were twenty-six with one death (a doubtful case). But what about the unvaccinated? In Middlesborough the children had been generally well vaccinated, but there were sixty-two such unvaccinated children attacked, and twenty-nine died. In Gloucester there were said to be 9000 unvaccinated children when the epidemic broke out, but fortunately some 8000 of them were hastily brought to the vaccinator, the conscientious parents thinking it wiser to throw their theories overboard. But 513 unvaccinated children fell victims to the disease, and 215 of them died, whilst many of those who escaped with their lives were hideously disfigured and some were blinded. Thanks to vaccination, smallpox has become increasingly rare among children, but where vaccination has been neglected smallpox, when it comes, is just as dreadful now as it was a hundred years ago, and just as fatal.

In East Africa the disease is very prevalent, and in Abyssinia it wrought frightful havoc among the troops under Ras Makonnen in 1898. But when the well-vaccinated Italian troops came into contact with the Abyssinians in the campaign of 1895, they escaped without a single case of smallpox, though they suffered severely from the other diseases from which they had not been protected.

Smallpox used to be extremely prevalent in Egypt and the surrounding countries. In the Sudan, in 1885, during the reign of the Mahdi, it is said that no less than 25,000 persons died of it in the town of Khartum alone, and the Bishareen tribe of Arabs are said to have lost a third of their number from smallpox in 1889. But in Egypt, where smallpox is so well known that there are no anti-vaccinators, proper vaccination is carried out fairly systematically, and thus, in spite of the constant importation of cases from these outlying tribes, the disease is not excessive.

Vaccination, even partially carried out, has diminished the extent of smallpox rapidly and to such a remarkable degree that it would be hard for anyone, who has not studied its history, to believe in its former prevalence and its horrors. If the universal adoption of vaccination and revaccination could ever be achieved, which is an ideal we all should have before us, there is no reason why smallpox should not be permanently and completely stamped out. We have seen this ideal almost attained in Germany. A little more science, a little less prejudice, would make it a comparatively easy matter in Great Britain. Unfortunately legislation on vaccination in this country in recent years has been exclusively in concessions to the anti-vaccinationists. The people among whom they agitate know nothing of the scientific basis

of vaccination, and readily believe the statements of the anti-vaccinationists that the decline in smallpox is due to improved sanitation and that vaccination is a dangerous and disgusting practice. I hope I have at least partially succeeded in these lectures in showing you not only the baselessness of such fears, but the uselessness of depending on hygiene alone to save us from smallpox. Do not be misled into the belief that isolation of cases is a sufficient safeguard against the spread of the disease. Dr. Werner has told us of the attempts in that direction made in his country before compulsory vaccination was introduced: "Isolation was most rigidly enforced; the patients were removed to smallpox stations and special houses; even seals were placed on the doors of the patients' rooms. The patients' clothes were disinfected or burned, and themselves and their attendants kept under watch and ward. All to no purpose; the pest found a way through the closed doors till suppressed by vaccination without the co-operation of sanitary measures." It is most earnestly to be hoped that England will wake up to the great danger to which she is exposing her people before it is too late, and we have once more to witness the unspeakable horrors of a smallpox epidemic such as those that swept across this country of ours in the dark days before Jenner.

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Lane's Conception of Chronic Constipation and its Management.—A. B. Cook considers that the two chief pathological factors concerned in the production and maintenance of chronic constipation are enteroptosis and acquired mesenteries or adhesions. These adhesions do not result from inflammation, but are developed to oppose the displacement of viscera, the tendency to which exists whenever the erect posture of the trunk is assumed. This is the basic feature of Lane's theory. The gross changes produced in the several portions of the large intestine are consecutive. The symptoms are those produced mechanically by the changes in the gut, and those due to absorption of toxic elements from the retained matter. According to Lane's teaching a proper conception of diseases of the appendix, gall-bladder, stomach, duodenum, pancreas, kidneys, ovaries, etc., in many cases requires that they be regarded as sequelæ of chronic constipation. In no circumstances should operative interference be contemplated till the surgeon has satisfied himself that every means of treatment has failed, whether medical or mechanical. In the milder type of cases in which non-operative means have failed to afford relief, the division of all the constricting bands and adhesions, together with careful attention to the bowel function, may prove effective. In the severer cases more radical surgery will be required, and this consists in dividing the ileum three to five inches from its junction with the cæcum and anastomosing the proximal end with the sigmoid or upper rectum, thus short-circuiting the fæcal current and eliminating the possibility of further toxic absorption. When pain is a prominent factor it is often necessary to remove the colon in part or in whole, preferably the latter. Cook believes that Lane's conception of chronic constipation and its management will be found to be the most nearly correct in principle and practice of all the numberless "new views" with which the literature of the subject is burdened.—*Medical Record*, vol. lxxviii, No. 8.

Spinal Analgesia.—Von Arlt considers that untoward results of intra-spinal injections are of a character best attributable to mild infection. In other words, we do not take sufficient pains to render the operation aseptic. His argument is based in part on the fact that whatever analgetic is used the unpleasant collateral effects and sequelæ are invariably of the same character, despite the chemico-pharmacological differences in these substances.—*Medical Record*, vol. lxxviii, No. 8.

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CONTENTS.

PAGE

***A Clinical Demonstration.** By PURVES STEWART, M.D., F.R.C.P. 353

***On Pneumonia.** A discussion by Sir JOHN BROADBENT, Dr. WEST, Dr. R. MILLER, Dr. CAMAC WILKINSON, Dr. THEODORE WILLIAMS, and Dr. G. A. SUTHERLAND 357

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A CLINICAL DEMONSTRATION AT THE POLYCLINIC.

By PURVES STEWART, M.D., F.R.C.P.,

Physician to Out-patients, Westminster Hospital; Physician to the West-End Hospital for Nervous Diseases, and to the Royal National Orthopædic Hospital.

GENTLEMEN,—There are some diseases which you recognise by means of complicated instruments, such as the microscope, the ophthalmoscope, or the cystoscope. I show you an example of a disease in which you want only one instrument—a telescope. Look at this man as he walks. Notice particularly that he does not swing his arms. That is an important diagnostic point. The ordinary person walks, more or less, with a swing. In the early stages of this disease the patient loses the swing in one arm, whilst the other still goes on swinging. When this man came in he had no swing of the arms at all. He will tell you that he has also a difficulty in using the left hand. Then you will notice, too, what an expressionless face he has. His only other symptom is cramp in the left foot. This is a symptom which is often found in paralysis agitans. As victims of this disease walk along they frequently feel cramp in their toes. Observe that this patient has no tremor. He is suffering from paralysis agitans *sine agitatione*. But even in the absence of shaking he has what is called the "interosseal" attitude of the hands. His thumbs and index fingers are adducted, his wrists slightly dorso-flexed, and the whole of the fingers bunched together. Many of these cases run their entire course without tremor, and this is an example, as I have said, of paralysis agitans without any agitation.

There is one other point. If you feel his pulse you will notice what a singularly low blood-pressure he has. One finds this in many cases of paralysis agitans. His blood-pressure is that of a young man; it is subnormal. In the absence of arterial

disease the blood-pressure in youth is usually about 120 mm. of mercury, and in middle age it rises to about 150. This man is æt. 51 years, and yet his blood-pressure is only 90 mm. of mercury. I have been noticing the blood-pressure in a number of cases of paralysis agitans, and the vast majority of them have this subnormal blood-pressure. I do not think this point is stated in any of the text-books, but it seems to me one of the outstanding features of the disease, and perhaps gives us an indication for treatment. Unfortunately the treatment of paralysis agitans hitherto has not been very satisfactory. We can only alleviate the symptoms by means of a few drugs, of which the most important is hyoscine. If, in addition, we give drugs so as to raise the blood-pressure, we can sometimes increase the effect of the hyoscine. I have been trying for some months the effect of pituitary extract in paralysis agitans in combination with hyoscine. In certain cases it has been very successful, and in others the result has been absolutely negative. The ordinary dose is 2 gr. In one patient we began with 2 gr. three times a day, then after that 4 gr., and though he has recently been getting 12 gr. a day, his blood-pressure has not gone up. He started with a blood-pressure of 120, and it is still there. Some patients seem to be resistant to the effects of pituitary extract, at any rate when given by the mouth. In other cases where it has had effect the tremor became less, and they did not feel so much lassitude and tiredness as they felt before. In an almost hopeless disease like this it is a useful thing to have another therapeutic weapon up one's sleeve.

Here is a man, æt. 35 years. He came in complaining that he had loss of feeling in the right side of his face, preceded by what he called neuralgia, which, however, has passed off. This was something like fourteen months ago. He began to have tingling in the right side of the face, and then he noticed that his face was numb. He complained of nothing else. His pupils, you will find, react normally to light, his external ocular movements are normal. Notice particularly that his sixth and seventh nerves are normal. Testing with a pin, you see that he has total anæsthesia and analgesia of the right side of the face, including the tongue, the nostril, the conjunctiva, and also the right half of the scalp in front. He has typical anæsthesia, complete, of the fifth nerve. When he bites his teeth his masseters contract uniformly on both sides, and therefore you would be inclined to think that the motor root of his fifth nerve is normal, but it is not. When he opens his mouth wide it swings round to the right side; it opens sideways owing to paralysis of the internal pterygoid on the right side. His tongue appears to deviate to the right, but that is because the jaw deviates, carrying the tongue with it. The patient has no anæsthesia anywhere else; his reflexes in all the limbs are normal and equal on both sides. He has the ordinary knee-jerks and ankle-jerks. He has no physical signs anywhere excepting palsy of the fifth nerve.

But, in addition, he gives us a curious history. One day, some ten months ago, when washing his face in the morning, he suddenly felt a twitching in one of his hands, in the ring and little finger of his left hand. The twitching spread up to his left shoulder, and he became unconscious. He evidently had a Jacksonian fit of his left extremity.

In a case like this we have to consider, first, where is the lesion? He must obviously have an organic lesion in the region of his fifth nerve. Is it inside or outside the pons? It is outside the pons, for this reason: that none of the other cranial nerve nuclei are affected, and none of his long cortico-spinal tracts are affected. If he had a lesion in the fifth nucleus he would almost certainly have had some signs of hemiplegia in the opposite leg or arm, from affection of the pyramidal tract. The lesion seems to be localised to the fifth nerve both motor and sensory. But could this lesion at the base produce a Jacksonian fit, commencing in the left arm? No. There must be another lesion, somewhere in the region of his precentral convolution in the right side.

What is the most likely cause, in a young man of this age, of an irritative lesion on the surface of the brain? The most likely cause is syphilitic disease. Although the patient denies syphilis, he admits gonococcal infection twice, once ten years ago and again two years ago. He denies any venereal sore. The probabilities therefore are that he has two syphilitic patches on the surface of his brain—one on the surface of the pons, probably in the meninges there, and the other on the surface of his right precentral convolution, probably in the meninges there. With a view of attacking the syphilitic origin of these two patches we have

given him mercury and iodide of potassium, but in spite of that his lesion has not improved. It has remained stationary. This does not contradict our diagnosis of syphilitic disease. If a gumma destroys nerve-cells or nerve-fibres no amount of mercury or iodide of potassium will replace the structures which have disappeared. All we can do is to remove the gummatous tissue and prevent the lesion from getting worse. The condition of the patient has remained stationary since we have had him; he is not any better, for part of his fifth nerve is destroyed.

Here is an interesting case from a diagnostic point of view. This is a patient with regard to whom some of you, possibly, will dispute the diagnosis which I am about to give. He is a man, æt. 44 years, and he states that about six months ago he suddenly felt as if something had got into his left eye, and then he saw double—that is to say, he suddenly had diplopia. He also felt giddy, but the giddiness passed off. The double vision, however, continues. Otherwise he feels perfectly well. When you come to look at him you will notice that the pupils are unequal, the left pupil is slightly larger than the right, and it is not absolutely circular when looked at with a magnifying glass. Then if you shade each eye alternately you will find that the pupils do not react to light. He has Argyll-Robertson pupils. His optic discs are normal, and so are all the other cranial nerves, with the exception of paresis of the left external rectus. He has no sensory change in the way of anæsthesia or analgesia of his face, trunk, or limbs. His motor functions are normal; he has no ataxia; his knee-jerks and ankle-jerks are brisk. In fact his only physical signs are the Argyll-Robertson pupils and the paresis of the left sixth nerve. As you will notice, he has brisk knee-jerks and ankle-jerks. This man, I believe, has tabes. I do not say that he has locomotor ataxia. Locomotor ataxia is a comparatively late stage of tabes. He has tabetic degeneration of his posterior columns. How do we know? Well, in the first place, he has Argyll-Robertson pupils. That is a condition which occurs in only two diseases—tabes and general paralysis of the insane. These are essentially the same disease, and they may be combined in the same patient. This particular patient has Argyll-Robertson pupils, which means that he is likely to have either tabes or general paralysis of the insane.

Some people might say that that is not enough to make a diagnosis of tabes from, because the patient has no sensory changes, and his motor functions and deep reflexes are normal. Well, we wanted some corroborative evidence, and with that object we examined the cerebro-spinal fluid, and we found it loaded with lymphocytes. This patient has marked lymphocytosis. Ten years ago we would have said, "This man may become tabetic." But to-day, because of the condition of the cerebro-spinal fluid, we diagnose tabes long before he has lost his knee-jerks or ankle-jerks. Why is he not a general paralytic? Because he has developed no mental symptoms. There is no organic disease excepting tabes and general paralysis of the insane which is associated with such marked lymphocytosis.

The last case we shall look at to-day is that of an intelligent little boy, æt. 9 years. Last year this boy was climbing a tree in Regent's Park, and he fell off the tree from a height, his mother says, of about thirty feet or forty feet. The park-keeper picked him up, and the boy was taken to hospital. They did not keep him in, so I think one may take the statement of a thirty-feet or forty-feet fall *cum grano salis*. The mother's estimate of the height is probably just as little accurate as an ordinary policeman's estimate of the speed of a motor-car. The chances are that this boy did not fall so much as thirty feet, but, in any case, he fell some distance and injured his head. Before then he was an ordinary healthy boy. His mother tells us that about a month after his fall he walked away to Harrow and came home next day. Since then he has done the same sort of thing twelve or fourteen times. He has walked out into the wide world and disappeared. The longest time he has been away is four days, and he was then found by the police in a state of exhaustion and semi-starvation. He always walks north. After a day or two he discovers himself in a field or some other extraordinary place, miles away. He walks sometimes for days together and sleeps out, and does not know why. He is brought home by the police, and after a few weeks dashes off again.

This is a case of what is called ambulatory automatism. There are two varieties of this disease—the hysterical and the epileptic. Which variety is it in this case? It is sometimes difficult to distinguish between the two. There are cases in

which we have no physical signs of epilepsy at all; there are other cases in which we have signs of epilepsy, generally of the minor or *petit mal* type. The mother of this boy states that at times he turns very pale and becomes excited; he stares and gets very bad-tempered, then suddenly becomes quiet. He never falls down, but sometimes he seems to be giddy, and walks round the room touching things and talking to himself very softly.

Well, now, that is quite sufficient to found a diagnosis of *petit mal* or minor epilepsy upon. The history of the head injury is corroborative. There is no centre in the brain especially associated with ambulation, but there is no doubt that this boy must have had an injury somewhere to his brain. We have examined the head and found no scar upon it, at which we were disappointed, because if we had found a scar we should have been justified in operating on that part of the skull in the hope that there might be some cranial depression or subjacent lacerated patch. The fact that there is no head scar also supports our view that he did not fall thirty feet or forty feet. But the result of the fall has been to produce a certain amount of cerebral disturbance, which has made this boy a subject of epilepsy of the minor type, followed by ambulatory automatism. It is much less common to have major epilepsy associated with automatism.

I have seen a number of cases of automatism, some of them very remarkable. One was in a South African financier. He was very hot-tempered, but we thought nothing of that in itself. One day he went to a board meeting in Johannesburg, and proposed a resolution at that meeting, and being a dogmatic sort of man and chairman of the company, carried that resolution. The carrying of the resolution necessitated an entire change in the policy of the company. On coming from the meeting he met a friend, and they went to lunch together. On getting back to his office he asked his clerk where he (the patient) had been. The clerk told him, and the employer said, "For Heaven's sake call another meeting for to-morrow!" He realised that he had lost recollection of something like four hours of that day: they were a blank to him. And yet he had been a normal business man, a man who conducted important transactions, some of them very complicated. He came home to England, and on inquiry I found that he was liable to sudden attacks of pallor, and once or twice he had bitten his tongue in his sleep. As time went on he developed major epilepsy. There could be no doubt that this was a case of epileptic automatism, and as the major epilepsy became more evident the automatism became less marked, and now he has no automatism, but ordinary major epilepsy. We usually get automatism in patients who have very slight fits. One classical description of automatism is that of a judge, who used to get up in his Court of Justice and micturate in public. Just before such an automatic action the person usually has a transient flash of unconsciousness, during which he does not fall down. Then he may begin to wander about, or to undress himself, or do other extraordinary things, and then he suddenly becomes conscious again. In the case of that financier we have the epileptic variety of automatism, the variety in which the person cannot revive the recollection of the lost day, or the lost hour, or the lost week. In a case of hysterical automatism, on the other hand, we can recover from the patient the whole history of his wanderings by hypnotising him and then questioning him. You can differentiate between the two varieties in that way. Of course we should always look for the presence of epilepsy as well, because the occurrence of hysterical automatism does not exclude epilepsy, nor *vice versâ*.

There was one case which got into the newspapers a few years ago, and was referred under the head-lines—"Have you seen this man? The man who has forgotten his life." This was a man who turned up at the Westminster Hospital, and said he had suddenly discovered himself on Westminster Bridge. The last thing he remembered before that was being in Waterloo Road some two years before. He also discovered that he had a new set of teeth which he did not possess before, and that he had a strange hernia and a truss to match. We found that he had scars on his head. He had been picked up on Westminster Bridge after a fit, with his tongue bleeding and his head cut. He also had a bottle of medicine with a Russian label on it, and some American money. He was rather a puzzle at first. We made out, of course, that he had *bonâ fide* epilepsy. When the man lost consciousness two years before, he was a cook, and he had evidently been abroad in the interval. He was subsequently hypnotised by another physician, and catechised as to what had really transpired. The history, which was afterwards corroborated, was that this man had a fit in Waterloo Road, that he fell down, and when he came round he engaged himself as a seaman on board a ship going to America. He made several voyages there. Then one day he came back to Glasgow, and deserted his ship there (possibly after

another fit), and re-shipped in another ship which went to Russia. He afterwards made a voyage to Australia. There he was involved in a street row and lost his teeth, and he bought a new set of teeth and came home. On the voyage he developed a hernia, for which he bought himself a truss after reaching home. He had another fit on Westminster Bridge, and woke up again, and to him these two and a half years were a blank.

It is an open question how much of that man's automatism was hysterical and how much was epileptic. The fact that he could recall his history under hypnotism rather suggested the hysterical type. On the other hand, he was also undoubtedly an epileptic, because he came in with a bleeding tongue and scars on his head. So that we have to face the possibility of the hysterical and the epileptic types separately, and the possibility of both combined. The boy we have seen to-day seems to be a victim of organic or epileptic automatism. We are giving him bromide, but he still goes on having these attacks. If we could find a definite scar on his skull we would seize upon that and have a piece taken out of his skull, and see if we could not find something underneath. But we do not feel justified in opening his head simply on an off-chance, because his case may be nothing more than the result of a general shake-up of his brain.

September 12th, 1910.

ON PNEUMONIA.

A DISCUSSION* BY

Sir John Broadbent.	Dr. Camac Wilkinson.
Dr. West.	Dr. Theodore Williams.
Dr. R. Miller.	Dr G. A. Sutherland.

Sir John Broadbent said: In a paper on "The Pneumococcus Infections" Professor Osler drew attention to "the enormous increase in the death-rate from that most important of pneumococcus infections, pneumonia," and emphasised the necessity for further study of this disease, more especially in relation to preventive measures. Amongst other points he raised as worthy of consideration were, the question as to whether a septicæmia precedes the development of a local lesion, and the question of the frequency and variety of clinically atypical forms.

I have thought, therefore, that it would be of interest to bring before the Society some cases bearing on these two important questions.

* Before the Medical Society of London.

The atypical cases are in adults, I think, met with chiefly in association with influenza. The insidious onset, the delay in the appearance of physical signs, the intermittent pyrexia may at the outset render the diagnosis obscure, while the absence of a definite crisis, the continued pyrexia lasting, it may be, some weeks, with progressive loss of flesh, will render the prognosis a matter of considerable difficulty, as in some cases empyema or malignant endocarditis may develop, whilst in others, after a prolonged period of septicæmia, recovery takes place without any further complication.

In children the tendency in apical pneumonia to the supervention of symptoms suggestive of cerebro-spinal meningitis may cause undue alarm, or the vagaries of a prolonged attack of lobular pneumonia may lead to a suspicion of pulmonary tuberculosis. Broncho-pneumonia complicating enteric in children may also be misleading, and mask the symptoms of the latter affection, which, in children, often runs an atypical course. For instance, I recollect one case in a child admitted to St. Mary's Hospital in which the irregularity of temperature, from the presence of broncho-pneumonia and the absence of spots or any classical signs of enteric, led to an error in diagnosis, and it was not till a typical relapse, accompanied by the appearance of typhoid spots, ten days after the temperature had fallen to normal that it was recognised that the initial illness had been enteric complicated by broncho-pneumonia and not the latter affection alone. I do not, however, propose to discuss further the difficulties in prognosis in atypical pneumonia in children, as these will be dealt with by Dr. Miller in his paper.

I will now proceed to lay before you brief notes of three atypical cases of pneumonia which occurred during a severe epidemic of influenza, in which the temperature was extremely irregular throughout. No crisis occurred, and the pyrexia was prolonged, lasting in one case as long as five weeks before recovery took place.

The first case was that of a man, æt. 42 years, admitted to St. Mary's Hospital on April 24th, 1900, under Dr. Lees, who has kindly given me permission to make use of this and the following two cases. He said he was at work till April 21st and felt quite well on going to bed on the 20th. He woke up on the morning of the 21st with bad

headache and was perspiring freely. In the afternoon he had severe pain in the left side of the chest, worse on taking a deep breath. He became very short of breath in the evening and could not sleep. He stayed at home on the 22nd and 23rd, and was admitted on the 24th to St. Mary's Hospital.

On admission he was a well-nourished, strong, well-developed man; lips cyanosed; pulse 126, respirations 42, temperature 104° F. There was dulness over the left base behind up to the angle of the scapula, with absence of vocal resonance and fremitus, and very poor air-entry. A few pleuritic crackles were audible over the back. There was dulness in the left axilla, with bronchial breathing in the upper part and bronchophony. The tongue was coated with a thick white fur. He was very restless and delirious.

On the 26th there was dulness over the whole of the left base in front and behind, with bronchial breathing over the back as well as in the axilla.

On the 27th there was dulness also over the lower lobe of the right lung, with deficient air-entry, and bronchial breathing was audible over a small area in the axilla. The temperature was extremely irregular, the pulse rapid and feeble, and the loss of strength very pronounced. He was very delirious throughout. The physical signs remained much the same to the end, and he died on May 4th—on the twelfth day after admission to the hospital.

At the autopsy the lower lobe of the left lung was slightly adherent to the chest-wall by a thick layer of sticky, recent lymph. It was intensely congested and œdematous, dripping with blood, soft to the touch, and plum-coloured on section. Its condition did not in the least resemble that of the red or grey hepatisation usually met with in fatal pneumonia. Portions cut out just sank in water. The right lung was also intensely congested, and the lower lobe presented the same characteristics as that of the left lung. Sections showed extreme congestion, numerous hæmorrhages, and partial collapse, with very little inflammatory reaction.

The pathological findings in this case are interesting, as throwing some light on the probable condition of the lungs in the two following cases, in which the pneumococcal infection took the form of a profound and prolonged septicæmia from the outset. It would appear that in this group of cases, though the lung was primarily attacked, the absence of the usual inflammatory reaction which occurs in typical pneumonia allowed of the pneumococci to flourish in a suitable nidus of congested tissue for an indefinite period, so that instead of the affection terminating by crisis, a prolonged septicæmia resulted.

The second case was that of a boy, æt. 14 years, admitted to St. Mary's Hospital under Dr. Lees on March 16th, 1900, for pain in right side of chest. He said he was well till the 13th, three days before admission, when he felt ill and shivery, and had a troublesome cough. On the 14th he had a rigor and severe pain in the head, and felt so ill that he went home, and then stayed in bed till the 16th, when he was admitted to St. Mary's.

On admission the pulse was 132, respirations 48, and temperature 100° F.

There was dulness in the left axilla, with pleuritic crackles, and dulness behind with deficient air-entry and friction-sounds over the base. On the right side there was a patch of dulness just below the angle of the scapula, with tubular breathing, bronchophony, and impaired resonance, with deficient air-entry behind. The tongue was coated with a thick white fur.

Both bases became completely dull behind, and tubular breathing developed over the dull area. The dulness and tubular breathing over both bases and the pyrexia persisted for four weeks, and the boy lost flesh rapidly. An exploring needle inserted on three or four occasions failed to discover any pus. Tuberculosis was then suspected, but no tubercle bacilli were found in the sputum. At length, on April 27th, after six weeks, the temperature fell to normal, and remained normal thereafter. He then put on flesh rapidly and gained strength, and was discharged cured on May 7th, ten days later.

The third case was that of a man, æt. 24 years, admitted to St. Mary's Hospital on May 4th, 1900, under Dr. Lees. A fortnight before he had felt ill, but kept on with his work till two days before, when he felt so ill he could not get out of bed. There was no history of rigors or pain in the side.

On admission the pulse was 96, temperature 101° F., respirations 28.

The patient was a well-nourished man, slightly cyanosed. There was dulness in the right axilla and over the right lower lobe behind, with deficient air-entry and pleuritic crackles. Tubular breath-

ing was heard on auscultation over the dull area in right lower lobe behind, but not in the axilla. There was also a small area of dulness at the base of the left lower lobe, with pleuritic crackles and bronchial breathing, which very soon cleared up. The tongue was coated with a thick white fur.

The dulness over the right lower lobe, with the tubular breathing and the pyrexia, persisted for nearly five weeks, when the temperature fell to normal and the patient rapidly regained flesh and strength. On his discharge on June 23rd there was still a dulness at the right base, with poor air-entry and deficient vocal resonance. There was also a small area over which bronchial breathing was audible, but the patient seemed quite well and strong, and the temperature had been normal for a fortnight before he was discharged.

These three cases illustrate the difficulties that may be met with in prognosis in atypical cases of pneumonia. The temperatures throughout were of an irregular type, and suggested the possibility of empyema, but the repeated exploratory punctures in the cases that recovered and the post-mortem examination in the fatal case negatived this. From the pathological evidence in the fatal case it would seem that in all probability there was little reaction on the part of the tissues, and the collapsed and congested lung formed a suitable nidus for the growth of the pneumococcus, which thus gave rise to a general septicæmia of prolonged duration.

In the following case, on the contrary, with a similar irregular temperature chart, an empyema rapidly developed. Edward J—, æt. 17 years, a porter, was admitted to the Hampstead Hospital under my care on August 13th, 1909. The patient stated that he was well till the morning of August 13th, and was filling a coal bucket at a flat when he felt ill and shivering, had a bad headache, and vomited. He went home, and felt very drowsy; was sick again in the evening, became feverish and thirsty, and complained of pain in the right side. There was no cough. He was seen by a doctor, who sent him into the hospital as possibly a case of sunstroke.

On admission he was a well-nourished youth, face pale, with cheeks flushed. Pulse 136, respirations 32, temperature 104° F. He was drowsy, and somewhat delirious at night. The chest was resonant all over on both sides. There were no adventitious sounds. The tongue was thickly coated with white fur. The abdomen was normal.

The next day, August 14th, the temperature was 101° F. in the morning, and the patient seemed better. Respirations 48, pulse 104. Resonance was normal on both sides, and there were no adventitious sounds. On August 15th the temperature was normal in the morning, but the pulse was still 100, with respirations 36, herpes being present on the lips and at the angle of the mouth. There was slightly diminished movement of the right side of the chest, and diminished air-entry. Resonance was slightly impaired over the right base. There were no adventitious sounds. The temperature rose to 101° F. in the afternoon, and the patient became very restless and delirious.

On August 16th the temperature was 98·2° F. in the morning, with pulse 80, and respirations 36. There was dulness over the right base behind, below the angle of scapula, extending forwards only as far as the posterior axillary line. There was impaired vocal resonance and fremitus over the area, with marked diminution of air-entry. Just below and slightly external to the angle of the scapula there was a small patch about 2 in. by 2 in., over which definite tubular breathing was heard with bronchophony. No crepitations were audible. Over the front of the right lung there was high-pitched skodaic resonance.

On August 17th the temperature was 99° F. in the morning, with pulse 84, and respirations 32. There was a cough, with expectoration of some blood-stained mucus. The physical signs in the lungs were not appreciably altered.

On August 19th there was dulness and tubular breathing over the upper part of the right lung behind, with increased vocal resonance and fremitus. There was dulness and poor entry, with absence of vocal fremitus and vocal resonance below the angle of the scapula as far as the mid-axillary line, where crepitations were audible. The temperature was 100·2° F. in the morning, 101·4° F. at night.

On August 23rd the patient seemed better. The tubular breathing over the back of the upper half of the right lung had gone and was replaced by moist sounds. Resonance over this area improved; there was no actual dulness, but complete dulness below the angle of the scapula behind, with absence of vocal fremitus and vocal resonance.

On August 27th the right lung was clearing up, except at the base, where dulness and signs of friction persisted. Moist sounds were audible over the upper half and in the axilla, with good air-entry.

On September 2nd, as the pyrexia persisted and the patient was perspiring at night, it was thought probable that there was an empyema. An exploring needle was inserted by the house-physician in the right base, but he failed to obtain pus.

On September 3rd an exploring needle was again inserted on the same side two inches below the angle of the scapula and two inches external and drew off pus.

The empyema was evacuated by Mr. Clayton-Greene on the following day. The patient took the anæsthetic well. He was put back to bed and seemed to be all right, but ten minutes later he had a sudden syncopal attack, which proved fatal in spite of all efforts to restore him.

At the autopsy the heart and pericardium, brain and meninges were normal. The empyema had been thoroughly evacuated. The left lung was intensely congested, hæmorrhagic, and partially collapsed, but there was no pneumonic consolidation, and its condition very much resembled that of the lung in the fatal case mentioned before. The only explanation for his sudden and unexpected death would seem to be that the shock of the operation on top of the profound and prolonged septicæmia had been too severe for him to withstand in his enfeebled condition. In this case it was three days from the onset of the symptoms and pyrexia before any definite physical signs in the chest supervened, and it would appear to prove, as suggested by Prof. Osler, that pneumococcal septicæmia may precede the development of local inflammatory reaction in the lungs or pleura. The toxæmia in this case, as in the three preceding ones, was a predominant feature throughout. From the character of the pyrexia at the outset and the similarity of the physical signs when they developed I thought that this case was analogous to the three cases already described, but contrary to expectation an empyema developed.

In contrast to these cases, and illustrating the difficulties in diagnosis as well as prognosis, is a case of protracted pyrexia, which simulated in all respects an attack of influenzal pneumonia at the outset, but which eventually proved to be pulmonary tuberculosis.

James R—, æt. 32 years, was admitted to the Hampstead General Hospital under my care on July 13th, 1909. He was well and at work till a month before admission, when he lost his appetite, and had shivering attacks and some pyrexia, thought to be due to an attack of influenza. On July 8th he had some pain in the right side of the chest, worse on taking deep breath or coughing. He then developed a troublesome cough and brought up some rusty sputum, and at night his temperature rose to 105° F.

On admission to the hospital on July 10th he was pale and very short of breath. Temperature 101·2° F., pulse 72, respirations 28. There was dulness and tubular breathing over the right base behind from a line 1 in. above angle of scapula, the base extending forwards to the mid-axillary line. Over the dull area there was increased vocal resonance and fremitus.

On July 17th a friction rub developed at the left base, the condition of the right lung remaining much the same.

The irregular pyrexia persisted, and the patient sweated profusely at night. A needle was inserted into the right base on three occasions with negative result. The physical signs in the lungs remained much the same, and though a blood-culture taken on August 28th proved sterile, it was thought it might be a case of delayed resolution with pneumococcal septicæmia, as no tubercle bacilli were found in the sputum. As he continued to lose flesh, and the night-sweats and pyrexia persisted, it was thought it might be a case of acute pneumonic phthisis, especially as some crepitation became audible over the apex of the right lower lobe. Both Von Pirquet's and Calmette's reaction for tuberculosis were positive, which seemed to confirm the diagnosis.

He was then put out on the balcony all day in the open air as far as weather permitted, and improved very markedly, the temperature falling practically to normal during the first three weeks of October, while he gained steadily in weight. The weather conditions and hospital arrangements did not allow of efficient open-air treatment, and towards the end of October the temperature again became irregular. On November 9th he was discharged after seventeen weeks in hospital. On admission his weight was only 7 st. 6 lb., but on discharge it was 8 st. 9 lb., and his general condi-

tion was much improved. There was still dulness and tubular breathing over the base of the right lung as far as the mid-axillary line, with a patch of dulness in the left axilla and a few crepitations at the apex of the lower lobe. He went down into the country to Devonshire, and at the end of December reported that he was gradually getting worse. I have had no further news of him since.

I have yet one more case of pneumococcal septicæmia to bring before your notice in which empyæma, malignant endocarditis, and suppurative meningitis developed. The patient was a man, æt. 48 years, who was admitted to St. Mary's Hospital, August 26th, 1909, under my care while in charge of the wards for Dr. Phillips, with the history that on August 5th he was taken ill with shivering. fever, and pain in the chest. He saw a doctor, who told him he had pneumonia and must stay in bed, where he remained till date of admission to the hospital.

On admission he was a fairly well-nourished man, who looked very ill, the lips and cheeks being cyanosed. The temperature was 99·2° F., pulse 96, respirations 26. On the right side the breath-sounds were better than on the left. There was left-front highly pitched skodaic resonance. There was dulness over left back below the angle of the scapula, extending forwards to posterior axillary line. Over this area there was absence of vocal resonance and fremitus, and poor air-entry. Over the right lung resonance was normal, rhonchi and sibili being audible over front and back. The pulse was regular in force and frequency, collapsing in type, suggestive of aortic regurgitation. There was marked throbbing of the carotids. The apex-beat was not visible nor palpable. Cardiac dulness extended 1 in. to right of sternum and 1 in. to left of vertical nipple line. A soft systolic and diastolic murmur was audible in the fifth space in the vertical nipple line. The apex-beat was not palpable, the first sound being very poor. With difficulty, owing to bronchitic sounds, a soft diastolic murmer was heard just to the left of the sternum in the third and fourth spaces. No murmur was audible at the aortic cartilage in the second right intercostal space.

The temperature on the evening of the 26th rose to 103·4° F. On August 27th, as the physical signs were suggestive of empyema, an exploring needle was inserted over the base of the right lung in three different situations, but no pus was withdrawn. The temperature in the morning was 99·2° F., but rose in the evening to 103·8° F. Urine, specific gravity 1022 ; small amount of albumen. Leucocyte count, 25,000 leucocytes to c.mm.

The intermittent pyrexia persisted, and he got steadily worse, and malignant endocarditis was diagnosed. Cultures taken from blood drawn from the median basilic vein, however, proved sterile.

On September 1st he developed right hemiplegia with aphasia, which pointed to embolism, and he died the same night.

At the autopsy there was an empyema at the left base, with thick viscid pus. The left lung was congested, collapsed and hæmorrhagic. There was a large brittle fungating vegetion on the aortic valve, due to malignant endocarditis of recent origin. There was suppurative meningitis, with lymph over the vertex and the left side of the brain, chiefly over the Rolandic area. No embolism was found, after careful search, in the middle cerebral and its branches.

This was an interesting case, and illustrates three of the possible sequelæ of pneumonia.

On admission, from the physical signs in the left lung and pyrexia, empyema was diagnosed, but as a needle failed to withdraw pus after three explorations, presumably owing to the viscid nature of the pus, it was thought that the lung condition might be similar to that in the first three cases described, in which there was presumably a layer of lymph or thickened pleura with collapsed lung beneath. The presence of a diastolic murmur, with absence of any hypertrophy of the left ventricle, together with marked cardiac dilatation and septicæmia, led to the diagnosis of malignant endocarditis of recent origin, which the autopsy confirmed.

The onset of aphasia, with paralysis of the right arm, seemed to point to embolism of the left middle cerebral artery or one of its branches, but careful search failed to reveal any embolus, so that the hemiplegia was presumably due to the suppurative meningitis, the exudation being most abundant over the Rolandic area.

I think the above cases are not without interest as instances of "clinically atypical pneumonia," and as illustrating the septicæmic character that pneumococcal infections may assume even when

the lung is primarily attacked. The three cases first described, and others of the same type of which I have notes, occurred during a serious influenza epidemic, and it is possible that influenza may be in many instances responsible for the atypical septicæmic type of pneumonia.

The most important ætiological factor, in the light of the pathological evidence referred to above, would seem to be the absence of the usual inflammatory reaction on the part of the lung when attacked by the pneumococcus. This lowered resistance might well be due, in many instances, to an attack of influenza immediately preceding.

The collapsed and congested condition of the lung, with the thick layer of overlying lymph found post-mortem in the fatal case, tends, I think, to throw some light on the cause of the delayed resolution, such as occurred in Cases 2 and 3, and is met with in analogous cases of influenzal pneumonia.

Dr. SAMUEL WEST said, before calling upon Dr. Miller he wished to ask Sir John Broadbent what evidence he gave, if any, in favour of general pneumococcal infection. He did not gather that Sir John obtained any cultivation from the blood, and he was not clear what evidence the diagnosis rested on.

Sir JOHN BROADBENT replied that there was only one case in which the blood was properly examined, and pneumococci were not found in that. But it often happened, even in malignant endocarditis, that one did not get a culture at the first or second attempt, as the micro-organisms were not always present in the blood-stream, but only when washed off into it at intervals.

Dr. REGINALD MILLER said: In dealing with cases of pneumonia in children, it is not uncommon to find instances in which the temperature of the patient remains raised for an unduly long period, and which for this reason give rise to considerable anxiety on the part of the physician. Yet on such cases, treated collectively, comparatively little has been written. It was thought therefore that it might, perhaps, be of interest to this Society if a number of cases of pneumonia in children showing protracted pyrexia were collected, and an attempt made to separate them into various groups according to the factors which, on clinical grounds, appeared to be responsible for the prolongation of the fever.

In order to make such a collection, it seemed to me best to select from a number of consecutive cases of pneumonia all those in which there was protracted fever, and to ignore such extraordinary instances as have been from time to time put on record by various authors. Although, therefore, my list of cases compiled in this way must of necessity be incomplete, I hope that it is to some extent proportionately representative, and that this advantage will be judged to outweigh the disadvantage of omissions.

For this purpose Dr. Leonard Guthrie and Dr. G. A. Sutherland, my colleagues at the Paddington Green Children's Hospital, very kindly allowed me access to their records, and to them my thanks are due.

In order to equalise the annual variations in the severity of pneumonia, I have taken a period of five years (1904 to 1908 inclusive), and have collected all the cases of pneumonia which came under observation before the end of the febrile stage, and which were not associated with any of the acute specific fevers nor, so far as was recognisable, with tuberculosis. The total number of these was 234. In 17, however, the date of onset of the fever could not be traced with sufficient accuracy for my purpose, so that this paper is based upon the remaining 217 cases.

In separating from these the instances in which there was protracted fever, I have taken arbitrarily the end of the tenth day as my criterion, and have classed as cases showing protracted pyrexia those in which the temperature had neither fallen by crisis, nor had shown definite signs of descent by lysis by the end of the tenth day. Where, after an interval of several days, the temperature has again become raised, although the pneumonic signs are disappearing, this second rise of temperature has been disregarded, and the case classed according to the length of the first febrile stage. On the other hand, since I am not dealing with cases of protracted pneumonia alone, but of protracted pyrexia in pneumonia, all cases in which, from whatever cause, the temperature has not fallen to normal within the specified time, have been classed as protracted. I have left out of consideration transient and seemingly unimportant rises of temperature, and have endeavoured to collect only such cases as, by reason of their lengthy period of fever, may give rise to difficulty and anxiety.

Lastly, in order to avoid (as I hope) the controversial subject of the nomenclature of the pneumonias of childhood, I have divided the 217 cases into primary and secondary pneumonias, including in the former cases of lobar pneumonia and primary broncho-pneumonia, and in the latter such cases as appeared to be due to an extension of bronchitis. These two groups contained 126 and 91 cases respectively.

Of the 217 cases no fewer than 55 (25·3 per cent.) showed fever for a period extending beyond ten days. Such a percentage as this cannot but be regarded as too high, and is, I suppose, due to the fact that the longer the fever lasts the more likely is the child to be sent to hospital. The proportions of the protracted cases in the primary and secondary pneumonias showed little difference, being 26·19 per cent. and 24·17 per cent. respectively.

When the fever is prolonged beyond ten days the danger to life is considerably increased, the death-rate in the protracted cases being rather more than three times as great as in the unprotracted. This is shown in the following figures. Taking all cases together the death-rate was 15·20 per cent., being 11·11 per cent., and 20·87 per cent. in the primary and secondary pneumonias respectively. But while in the unprotracted cases only 9·87 per cent. died, in the protracted the mortality was 30·90 per cent.

When we study the fifty-five cases in which there is protracted pyrexia we find that they can be put fairly conveniently into five classes according to the clinical conditions which appear to be responsible for the prolongation of the fever. In five instances, however, the case comes under more than one heading.

The protracted pyrexia may thus be associated with:

(1) Protracted simple (non-spreading) pneumonia.

(2) Protracted spreading or recurrent pneumonia.

(3) Imperfect resolution.

(4) Bacterial complications other than pneumonic.

(5) Toxæmic complications.

Under the first two headings are included two thirds of the cases.

In the first group come those cases of pneumonia which do not differ from the ordinary except in that they run a course of over ten days. The fever for the last seven days is of the remittent type, and the crisis is preceded by a pseudo-crisis.

There are 16 examples of this class among the 55 cases with protracted fever which, with three exceptions, are primary pneumonias. The mortality in these primary cases is considerably above that of the unprotracted primary cases, being 23·07 per cent. instead of 5·48 per cent. The three cases of secondary pneumonia in this group all died. The great majority of the cases in this class came to an end either by recovery or death by the twelfth day of disease.

In the second group I have placed those cases in which the pneumonia spreads in the lungs from one focus to another. These are of common occurrence. In the 55 cases there are 23 instances of such a spreading, 17 in primary and 6 in secondary pneumonias. I regret that my list does not include any of those cases in which the pneumonia creeps about the lungs for a period of weeks. In the majority of this group only one second focus makes its appearance, in three the pneumonia spread twice, and in one, a case complicated by septicæmia, to be referred to later, three portions of the lungs were invaded in addition to the primary focus.

The death-rate in this group was 17·64 and 16·6 per cent. for the primary and secondary pneumonias respectively.

The second attack of pneumonia is as a rule shorter than the initial one. The pneumonic process may spread early while the fever is at its height, or later when the temperature is falling. Occasionally no fresh signs of pneumonia can be recognised, but the reappearance of the pneumonic symptoms, and the renewed pyrexia are very suggestive.

I will quote four examples of this variety. In one case the pneumonia spread from the right base to the left lower lobe, and on the nineteenth day of the disease the left upper lobe became involved, the child dying on the twenty-fourth day of heart-failure. In another patient, after a fall by lysis almost to normal the temperature rose again, with involvement of the right middle lobe, and ultimately fell by crisis on the nineteenth day of the disease. In the third patient, after an initial attack lasting for fifteen days the temperature again rose, and although no fresh physical signs appeared, the renewed symptoms and the course of the temperature with its critical termination left little doubt but that a fresh patch of pneumonia was the cause of the second wave of pyrexia. The fourth case showed the same points in a secondary pneumonia.

This spreading variety of pneumonia is most commonly found in connection with whooping-cough and measles, but none of my cases were associated with either disease.

In Group 3 the protracted pyrexia is associated with imperfect resolution, or with, at all events, the persistence of some of the physical signs in the chest. This is much less common, and includes only 9 of the 55 cases. All of these were secondary pneumonias. Only 1 of the 9 cases died.

The temperature in this variety may be irregular, but is often swinging, remittent, or intermittent in character. When we come to examine these cases we find there are no signs of any fresh patches of

pneumonia, but that the abnormal physical signs in the chest remain unaltered in position and are almost invariably at the base of the lung. There may be only the signs of slow resolution, but in 7 of the 9 cases the physical signs at the affected part of the lung became those of dulness and much diminished air-entry, giving rise to a suspicion of pleural effusion. In several, indeed, an exploring needle was put into the chest, but no fluid was found. In one patient the signs were those of dulness and poor air-entry during the second and third weeks of illness, but no exploring needle was used. In another patient, after a spread of the pneumonia from the right base to the right apex there arose a suspicion of a right-sided empyema, and the chest was punctured on the twenty-second and twenty-ninth days, both times with negative results. In other cases in this group the oscillations in the temperature are not so great as in the charts shown.

In such cases as form the majority of this group it would appear that doses of poison, large or small, are being absorbed, and the physical signs suggest that in most instances this toxin is derived from a thick layer of exudate upon the pleura.

In this connection I may refer to 3 cases not taken from the 55 cases I am considering. The first had the ordinary termination of a case of primary pneumonia in a child, the temperature running fairly evenly below normal, and the lung signs clearing up quickly. The next was a case of pleuro-pneumonia in a child, the physical signs suggesting a moderate amount of pleurisy, and here a swinging temperature was noticeable. When an empyema is developing, so much toxin is absorbed that the temperature usually quickly becomes high and sustained. The third case showed the swinging temperature again; it was a case of empyema which had been opened, but in which drainage became temporarily imperfect, although not entirely stopped. Syringing out the empyema cavity brought about an immediate drop in the fever. A similar swinging temperature is sometimes seen in cases of otitis media in which the ears are discharging.

It is perhaps difficult to imagine that such large oscillations of temperature can be due to absorption of toxin from a pleural exudate that does not develop into an actual empyema, but the physical signs and the course of the disease seem to suggest this explanation.

In Group 4 protracted pyrexia associated with bacterial complications other than those already mentioned is not common. Although all the conditions such as empyema, purulent pericarditis, infective endocarditis, otitis media, suppurative meningitis, peritonitis, septicæmia, pulmonary abscess, and others are included, only eight of the fifty-five protracted cases come into this group. The reason for this is that these bacterial complications usually either arise early and cause the death of the patient within a short time of the onset of the disease, or they develop after the temperature has been lowered for a few days, and so do not appear as causes of protracted fever in pneumonia.

One of these cases in which there was a streptococcal septicæmia is of interest. The patient was a boy, æt. 5 years, under the care of Dr. Sutherland. He was taken suddenly ill eight days before admission to hospital. When seen on the eighth day he showed signs of a right basal pneumonia, and during the course of the illness the pneumonic signs spread from the right lower lobe to the left lower lobe, thence to the right upper lobe, and, finally, to the right middle lobe. Streptococci were obtained from the lung on the thirteenth day, and from the blood on the forty-second day. Anti-streptococcal serum (polyvalent) was given on three occasions, but only once seemed to have a temporary beneficial effect upon the temperature. After fifty-four days the fever disappeared, and the boy gradually recovered.

The toxæmic complications constituting the fifth group of cases are of little interest. Four such are included here; three were the result of enteritis occurring in the course of secondary pneumonias of infants. The fourth case was one of acute nephritis, which I should have preferred to class as a bacterial (pneumococcal) complication, but which for lack of evidence is placed in this group.

Dr. Camac Wilkinson said he was chiefly interested in the first group of cases, because in the first place, in pneumonias occurring in children, it was not always easy, especially in cases which recovered, to work out the bacteriological element in them, as very often children with this condition did not expectorate. But in the first series of cases read there were many points worthy of notice. Looking at the temperature charts alone, one would be very loth to make a diagnosis of croupous pneumonia. When the term "pneumonia," meaning inflammation of the lungs, was used, it was capable of various interpretations. But if it were croupous pneumonia one was much surprised at the want of the ordinary pulse respiration ratio in two or three of the charts, and as no mention was made of the character of the sputum, he did not know whether the sputum had the character of typical croupous pneumonia at all. But a great deal of the interest in the series of cases brought forward by Sir John Broadbent was lost in the absence of evidence of the nature of the causal infective agent. In hospitals nowadays one should carefully analyse the expectoration by the most modern methods, and if one failed to find sufficient reason for, or evidence of, a disturbing element in the way of another organism, his conclusion practically always was that there was

tuberculosis complicating the trouble. Tuberculosis was one of the complicating features in these pneumonic cases, and even in the early cases tubercle bacilli might be found in the sputum. Otherwise, if one put aside those cases, it was of the highest importance to know whether the inflammatory condition in pneumonia was due to one or two or even three or four of the organisms which were associated with pneumonia—pneumococci, streptococci, influenza bacilli, staphylococci, or the so-called Friedlander's bacilli. Those factors had to be looked for so as to know what were the causes acting as disturbers of the temperature. Of all the cases he had seen exhibited that evening, judging from the temperature chart alone, unless those other factors had been brought forward—the disturbance of pulse-respiration ratio, the presence or absence of the typical sputum, and the presence or absence of the specific organism, he was bound to say he would hesitate to accept the diagnosis of croupous pneumonia. In the second chart the curve was very much what was called the streptococcal character, and in that case he thought probably a streptococcus was the essential cause, if only because it was rather a prolonged course, and the patient ultimately recovered. One assumed that the streptococcal pneumonias were less virulent and dangerous than the pneumococcal. The pneumococcus was one of the most acutely virulent organisms which could enter the human body. When one had no evidence of influenzal infection of a secondary character, personally he was not disposed to use the term "influenza." He recognised that there were other organisms which would produce a type of disease like that of influenza. The term "influenza," if one wished to be accurate in diagnosis, should be largely restricted to those cases, at any rate in lung conditions, which were due to the specific influenza bacillus, because by proper methods one could infallibly demonstrate the influenza bacillus in sputum as easily as the presence of tubercle bacilli in the sputum. And if the influenza bacillus was not proved to be present, one was at a loss to know, from the clinical standpoint, whether the disturbance was due to the influenza bacillus or not. In some of the cases it may have been. That was so in the first case certainly, because the description rather suggested influenza than pneumonia. But if the signs in the early stages were those of croupous pneumonia, he would be inclined to exclude influenza. He had seen cases of fatal influenza, but he had not seen the typical condition which one found in croupous pneumonia, but rather, as Pfeiffer described, those little areas of what looked like pus which could be squeezed out of the small bronchioles. With regard to the other important factor which might have been acting behind the scenes, he thought in one of them, if he had been asked, he would have said it was of the nature of tuberculosis. He did not think it was generally recognised how great might be the vagaries of tuberculosis as a complicating agent in those cases. He had a case of pneumonia in a boy, æt. 15 years, who got well and the pneumonia disappeared. After the normal course of pneumonic temperature and the fall subsequently, the temperature went up to 105° F., and with rigors. It fell next day. On five successive days it went up to that temperature and fell again. He thought the patient must have some streptococcal condition, perhaps some purulent condition, and he thought possibly it might be interlobar. He needled in several directions, but could find nothing to account for it. He gave two 10 gr. doses of quinine; the temperature came down and the boy apparently got well. A month later he tested that patient with tuberculin. At the first dose the temperature went up to 103° F., so that obviously in him the disturbing element was of a tuberculous character. Another interesting case was one of prolonged temperature extending over twelve weeks. The case was as follows: A maid in a doctor's house had a typical attack of pneumonia, affecting the apex—at any rate, beginning there and spreading downwards. The temperature was of the ordinary kind, and the sputum showed pneumococci, but no tubercle bacilli. There was no crisis. The temperature went on for twelve weeks, and during that time she had seen seven other doctors. Finally he (Dr. Wilkinson) was called in, and concluded that tuberculosis was operating somewhere. After she had gone on in that way for some weeks he suggested she should come into the hospital. As soon as her temperature became normal he gave her a test dose of tuberculin and her temperature rose to 103° F. At that time she was reduced to 4 st. 8 lb. in weight, having formerly been 8 st. In the course of three months' treatment by tuberculin she gained nearly 2 st. Four years afterwards, which was a year or so ago, she was back at the doctor's house working as formerly, and had gained another 2 st., in other words, had resumed her normal weight. Not only the reaction of that case to tuberculin, but the extraordinary result of treatment with tuberculin, left no doubt that she had tuberculosis. Another case was that of fever in a woman whom he was treating for two or three months with tuberculin: she was obviously in the first or second stage. She suddenly had a temperature of 104° F. At that time she was having treatment outside the hospital. He took her in and her temperature went up to 104° F. and continued to do so every night for ten nights, coming down to the normal in the morning. In that case he proved there was a mixture of streptococcal infection with the tubercle, and he gave her what he had found of so much benefit in those cases—one intra-venous injection of collargol; after twelve hours the temperature had come down

to normal; it remained so, and she was now perfectly well. Another case, of a different kind, was one of pneumonia, in which the temperature had been for eight weeks between 103° and 104° F. continuously. Then tubercle bacilli were found. In that case also he gave three intra-venous injections of collargol, and the temperature came down to normal and remained down until death. One lung was consolidated and half the other, but the patient remained alive six months, eventually dying from sudden hæmorrhage in a few minutes. All those cases showed clearly that unless one took means to find out whether tuberculosis was present one might lose sight of the most important cause.

Dr. THEODORE WILLIAMS said that he had seen similar cases to those mentioned by Sir John Broadbent, and had not always been able to come to a definite conclusion about them. He had intended asking the same question which Dr. West asked as to the proof of infection by micrococci. It seemed to him that they were not typical cases of that kind at all, and if he had been asked about those cases without going into such full details as had been given, he would have regarded them as examples of those curious cases which are seen after influenza, and sometimes proved fatal. He had seen cases in regard to which he would have given a favourable prognosis, but nevertheless many of them died in the most extraordinary and unexpected manner. Collapse had come on, and without the typical crisis, but there had been the peculiar temperature, which he described as see-saw temperature, and the case had gone to the bad and died, generally, from the failure of the heart's action. He was rather surprised that Sir John Broadbent had to test so often for effusion. He had seen such cases of effusion developing, but generally it had not been necessary to use the needle; the condition had been sufficiently clearly shown by physical signs. He could not agree with all the evidence in favour of tuberculosis which had been mentioned by Dr. Wilkinson. In one case he said he injected tuberculin, and because it reacted once therefore the case was tuberculous. Every now and then one obtained a single reaction from tuberculin in patients who were probably not tuberculous, and it was necessary to test many times before concluding a case to be tubercular. In many cases of pneumonia tubercle did appear as a complicating element, and this must be borne in mind. In many cases the patient was thoroughly depressed by the pneumonia, and a ripe prey for the invasion by the tubercle bacillus. The second paper was also very interesting, and he congratulated Dr. Miller on the great variety of the cases which he had recorded of infantile pneumonia. They were a difficult class because they varied so much, and the temperatures of children went up and down so much, and with such slight cause, that it was difficult to come to conclusions about them. But Dr. Miller's cases seemed to have done fairly well, and the mortality was not high. Dr. Miller's classification of cases was very good.

Dr. G. A. SUTHERLAND said, as had been already pointed out, it was very difficult to classify pneumonias in children. Dr. Miller classified them into primary and secondary. Some years ago he (Dr. Sutherland) looked over some of his cases, the diagnosis of which was marked on by the resident medical officer. He found a large number of them had been labelled catarrhal pneumonia or broncho-pneumonia which were certainly of the type he would himself prefer to call pneumococcal pneumonia, of the lobar type. He looked at the temperature chart, and there was seen to be a shorter or longer pyrexial period, usually under ten days, with a sudden crisis. On looking at the notes it was evident that there was probably one area of the lungs affected, and catarrhal signs elsewhere. He thought a temperature chart of that character, with a sudden crisis, showed the case to be definitely one of pneumococcal infection, and that was probably a better term than either catarrhal pneumonia or broncho-pneumonia. The last-named term he confined to those conditions which were secondary to infectious fevers. Clinically, there was no comparison between those cases and cases of pneumococcal pneumonia; the whole course of the illness and the symptoms all through were so different that, clinically, one could separate them very well. He did not know how Dr. Miller arrived at his classification of primary and secondary, though perhaps it would be more evident when reading the paper after it was published. From pure statistics of pneumonia in children he (Dr. Sutherland) would not be prepared to draw any definite conclusions because of the extreme difficulties in connection with their classification. One very interesting point struck him in connection with Dr. Miller's remarks, namely, his explanation of the zig-zag temperature after a crisis in pneumonia or before it. Dr. Miller suggested that in cases where there was increased dulness going on to flatness, in which one was induced to explore the pleura, there was an effusion into the pleura, and that from that an absorption of toxins took place. It was known from the complications in connection with empyema how absorption and infection proceeded very markedly. One also knew the frequency with which empyema led to pericarditis or meningitis of a suppurative character. Did Dr. Miller believe, as many believed, that there might be a simple non-purulent effusion, or whether it was always purulent? It was held that the majority of cases of purulent effusion would not be naturally absorbed. In the cases such as had been referred to, and of which he had seen examples, it sometimes seemed to him

that the mere act of putting the needle into the pleura or into the lung had a beneficial effect, and after a time the temperature improved. He did not know how it acted, but he believed the fact was familiar to many present. Did Mr. Miller think the zigzag temperature was always due to the presence of pus, or whether in some cases the same form of temperature might not be due to the presence of a mere serous effusion?

Dr. WEST thought general pneumococcal infection was a very difficult subject, and he was not ready to accept off-hand much of that which was written and talked about pneumococcal infections. He believed it was probable that they would get back to the same views that were now in vogue concerning streptococcal infections, namely, that there was not one, but were many infections to deal with. And he was led to that view largely because the statistics of the complications of pneumonia taken from the clinical side were so different from those which came from the post-mortem room only. He was inclined to attach more importance to the clinical rather than to the pathological evidence. He thought the matter was very far from being settled. One should not speak in a loose way of pneumococcal infection without having definite bacteriological proof of it, and that was not generally forthcoming, and secondly, that the general infection was due to the same pneumococcus as that which produced acute primary pneumonia. The evidence was very scanty which went to prove that pneumonia itself was but a local manifestation of a general infection. The evidence, indeed, seemed to point the other way.

In dealing with atypical pneumonias it was very much a bacteriological question; it could only be attacked satisfactorily from the bacteriological side, and such a method of research was very difficult in the case of the lung. Another point dealt with by Sir John Broadbent was the close connection of those atypical pneumonias with influenza. In drawing up statistics of pneumonia some years ago he tried to avoid that difficulty by taking statistics before the influenza time, because influenza vitiated the statistics in connection with what might be termed old-fashioned pneumococcal pneumonia. There were many reasons for that. He did not think it was proved, although some of the speakers had suggested it was, that many of the pneumonias were due to the influenza bacillus. It was, he believed, generally pneumococcal pneumonia after influenza. Influenza seemed to reduce the resistance of the body generally, and so that the pneumococcus could get entrance and grow just as measles and whooping-cough predisposed the one to the other. There could be little doubt that many of the pneumonias in atypical cases were mixed infections. Some of them might not be pneumococcal at all, although the acute inflammation suggested pneumococcal invasion. He thought it would be well to get back to the use of some such general term as "acute inflammation of the lung," which did not imply its bacteriological cause. When one spoke of primary and secondary pneumonias, one meant by primary idiopathic pneumonia generally, no doubt, pneumococcal, and the secondary as that which came on after bronchitis, which therefore might be termed post-bronchitic, and which might be of streptococcal or of some other origin. It was interesting to him especially to find the terms "primary" and "secondary" pneumonias in relation to broncho-pneumonia being accepted by degrees. The tubercle bacillus itself could excite an inflammation in the lung of such intensity as to resemble pathologically acute pneumonias, and clinically too. So often one found that a case which was apparently pneumonia to start with and looked like pneumonia, did not run the course of ordinary pneumonia, and ended in acute tuberculosis. He did not think it was true to say that that pneumonia became tubercular, which was the old-fashioned theory, but it was often from the beginning, as could be shown, tubercular. With regard to the result of the various injections, Calmette, von Pirquet, and even tuberculin injections, the mere fact of a reaction following did not prove that an inflammation was tubercular; it might perhaps prove that the patient had tubercle, but it did not prove that the particular inflammation was tubercular. But if there was a marked reaction in a patient who had acute pneumonia, knowing what one did, that in acute tuberculosis the reaction often failed, it would rather tend to prove that the inflammation of the lung was not tubercular. The difficulties in diagnosing those cases in many instances from typhoid, tuberculosis, septicæmia, malignant endocarditis, and even influenza were very great. Two or three of the cases, the charts of which were shown by Sir John Broadbent, might very well have been empyema, but he supposed that if they had coughed anything up it would have been stated in the description of the case. But those empyemata were sometimes very difficult to diagnose, and they were often not discovered until the patient coughed it up. Influenza in itself tended to make pneumonia run an atypical course. There had been much pneumonia about in the last year or two, which had run a very long course, although it had ultimately got well, perhaps lasting ten days or a fortnight, and then ending with a crisis. Another interesting point was in the remarks of Dr. Miller, viz. the irregular temperature in connection with the absorption of toxin from the lymph exuded on the pleura, but of which there might have been very little physical evidence. But he would like to put the other side, where, after pneumonia, pus was found in the pleura and even in the pericardium, too, without any rise of tem-

perature or anything else in the patient's condition generally to suggest that he had anything of the kind the matter with him. The patient might have pneumonia, get apparently well, be convalescent, sit up in bed eating his dinner, and then fall back dead, and he might be found to have a double empyema, pus in the pleura being of such amount that no one could have missed it if they had looked for it. If the toxins were going to produce symptoms in the one case, an explanation was wanted why they did not in the other. The points which had been brought forward in the discussion had been very interesting, and had raised others which perhaps future communications to the Society might elucidate.

Sir JOHN BROADBENT, in reply, said he brought the cases forward because they were to him at the time an extremely difficult group to classify, and presented great difficulties in prognosis. He had been much interested in the remarks of Dr. West as to the relationship of influenza to pneumonia. Most of the atypical cases had occurred after epidemics of influenza, and he much regretted that proper bacteriological examinations were not made of the cases at the time. The cases occurred in 1900, when there was a very severe epidemic of influenza, and so much attention was not then paid to the bacteriology of the condition as now. If that examination had been made, perhaps he would have been able to throw more light on the kind of case. But he agreed that it was rather that the influenza predisposed and lowered the resistance of the patient, so that the pneumococcus got in and set up an inflammation. He did not think it was the influenza bacillus itself which set up the prolonged septicæmia, and the consolidation of the pneumonic type. With regard to the question raised by Dr. Miller, as to whether the organisms flourished in the thick layer of lymph overlying the congested and collapsed lung itself, he thought it was doubtful. He had seen such cases in adults, and in those cases lung beneath the layer of lymph had not been in a state of pneumonic consolidation, but rather in a collapsed and congested condition. Dr. Sutherland raised the question whether it was pus or lymph, and it was a difficult point as to where to draw the line. In most of those pneumococcal conditions there was a thick layer of sticky lymph, like that seen in rheumatic pericarditis, not a diffluent soft purulent fluid. He had been interested in the classification carried out in Dr. Miller's paper. The first classification he, Sir John, ever got of pneumonias was from Dr. Samuel West's book. Previously it had always been a puzzle to him, because everybody had spoken of pneumonia in children as broncho-pneumonia, and it was obvious that many of those cases were not, in the strict sense of the term, broncho-pneumonia, but primary pneumococcal croupous pneumonia. The classification into lobular pneumonias and primary or secondary broncho-pneumonias, according to the ætiology, cleared the way for a much better understanding of the pneumonias in children. Another important question which was raised was as to the relationship of pneumonia and tuberculosis. Were those acute cases which set in with a rigor and signs of consolidation, and proved to be tubercular eventually, primarily so from the outset, or had a pneumococcal infection set loose tubercle bacilli which previously had been locked up in bronchial or other glands? It seemed to him that in measles, of which pulmonary tuberculosis was often a sequela, even in cases where there was apparently no chance of recent infection, the inflammatory reaction might, perhaps, set loose tubercle bacilli previously locked up elsewhere in the body. He thought that in cases of acute pneumonic phthisis something of the same sort happened. That was a very important investigation to follow out. In the spreading lobular pneumonias of children it was often difficult to determine for weeks whether a child would get well and whether the affection was pneumonia or tuberculosis. In these cases also there was the same doubt as to whether the case was primarily tubercular. Pulmonary tuberculosis did not generally start with a rigor and rapid consolidation, but was more insidious in its onset. Yet some of the cases which started so acutely eventually turned out to be tuberculous.

Dr. MILLER, in replying, said he was glad he had not attempted to discuss the ætiology and classification of his cases of pneumonia in children, because it was a matter of far too great difficulty. It was fairly clear from the discussion how it was that so few papers had recently been given on the subject, there being but little agreement on these points. In answer to Dr. Sutherland concerning the cases in which a swinging temperature developed after the pneumonia, namely, whether he regarded them as cases of serous effusion or purulent effusion, he feared he had not made himself clear on that point, because he inclined to regard some of them not as cases of effusion, but as cases in which, in the majority of instances, there was a thick exudate of lymph upon the pleura; Sir John Broadbent had dealt with that point in his reply. In the nine cases which fell into that group out of the fifty-five, seven had the signs of diminished air-entry and dulness amounting almost to flatness, and such physical signs would go very well with a thick exudate on the pleura. In one other case, however, it was definitely reported that the air-entry was extremely loud and the broncial breathing was remaining, and it seemed that in such a case the toxins must be accumulated in a lung in which resolution had not occurred.

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CONTENTS.

PAGE

*On Rectal Fistula. By FRED C. WALLIS, F.R.C.S.... 369

*The Early Diagnosis of Subacute Combined Degeneration of the Spinal Cord. By E. FARQUHAR BUZZARD, M.D., F.R.C.P. 374

*The Diagnosis and Differential Diagnosis of the Pathological Processes causing Enlargement of the Kidney. By CHARLES GREENE CUMSTON, M.D. ... 377

* *Specially reported for the Clinical Journal. Revised by the Author.*

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ON RECTAL FISTULA:

A DEMONSTRATION AT CHARING CROSS HOSPITAL.

By FRED C. WALLIS, F.R.C.S.,
Surgeon to the Hospital.

GENTLEMEN,—You will remember the other day I was talking about fistula. Now there is very little to see in this man, but you are able to see a brown pigmented condition over the buttock which is around the external opening of a fistula. By pressing along here I find that there is a hard swelling, which comes to the mid-line posteriorly, and I can feel an elongated area of induration like a pipe-stem. This means that the internal opening of this fistula is at or near the mid-posterior line, which is a little unusual, having regard to the position of the external opening. And this fistula, therefore, comes rather into the category of what we call "semi-horseshoe fistula." The external opening is two inches from the anal margin, and running towards the mid-posterior line is a subcutaneous, hard, chronic inflammatory area, feeling, as I say, rather like the stem of a pipe, which represents the track, and that track leads to the internal opening, which is situated at or near the mid-posterior line, and in the region of the two sphincters, either between them or at the margin of the junction of the proctodeum and the end of the gut. When we know what a fistula looks like we can build upon that knowledge. It is a most troublesome complaint. All these common rectal ailments, and this one particularly, arise from that region of the alimentary canal which is called the "ano-rectal region," which is situated just where the proctodeum joins the blind end of the gut. Some of you may have seen a new-born baby with imperforate anus. In such a case you will see a very thin membrane across the anus, which is intact and has not quite been absorbed. When that has been absorbed and the outlet is properly

formed, what happens is—that you have had a large bulging piece of gut which has had to fit on to a comparatively small proctodeum, and so there is a certain amount of puckering of this large blind end of gut, to be able to fit the small opening of the anus, and consequently you get an anatomical condition which is now talked much about—the crypts and columns of Morgagni. These are sometimes exaggerated, and sometimes the fusion is a rough piece of workmanship, and as the infant grows these grow in proportion, and then it may overgrow and there may be possibly a polypoid condition existing in these areas. When some hard fæcal matter comes down behind this it tears it, and there is a raw exposed surface. Another way in which the same thing can happen is that often there is a pocket running down, and sometimes a pocket which runs up a considerable way underneath one of these crypts of Morgagni. These also may, and do, lose their epithelial lining, and get bruised and possibly ulcerated, and cause a great deal of pain, such pain often lasting for hours. If this exposed surface becomes infected with the micro-organisms which are in the alimentary canal, there is every likelihood that suppuration will occur. Given a lesion there, you have all sorts of conditions of things which may happen with regard to inflammatory swellings arising from it. The pus-producing organisms may either track up underneath the mucous membrane, or else underneath the skin, or in the ischio-rectal fossa, or in the pelvi-rectal fossa. If this pus gets into the ischio-rectal fossa you will have a large swelling to start with, which bursts just outside, and then you get the external opening. You may get the pus coming up above the levator ani and invading all this area, causing a pelvi-rectal or a retro-rectal abscess. These are the suppurative conditions which may arise from a lesion of the ano-rectal junction, which is anywhere within an inch to one and a half inches from the anal margin. You do not want to make a high digital examination to find the reason for this lesion. It is always just within the anal margin, and that is why so many of these conditions are missed. It is a common idea that the proper way to examine a rectum is to examine it to the full extent of the finger, at which point you are not likely to find anything, as a rule, except a growth. All the common rectal ailments are found at or near the ano-rectal junction. The infection starting from this abrasion goes in one of five directions, either submucous, subcutaneous, ischio-rectal, pelvi-rectal, or retro-rectal. All these varieties of fistula, from which condition this man is suffering, are preceded by an inflammation and the formation of pus. An abscess will be more or less obvious and more or less distressing to the patient according to the existing tension. Take the submucous one. If there is a small outlet for the pus, what happens is that the abscess cavity gradually fills up, becomes distressingly painful to the patient, and at last the tension is such that it overcomes resistance and some of the pus escapes. Then the symptoms ameliorate, and the patient for the time being improves. But the infective material is still there, and the cavity fills up again, more pus comes, and when it is evacuated improvement occurs and relief follows again. This goes on until there is a large area of suppuration not only in length but around the circumference of the bowel. That is the way in which these abscesses form, relieve themselves, and recur. The submucous one is the form in which it occurs most. But when you have a suppuration into the ischio-rectal fossa there is a very large area in the ischio-rectal anatomy where large collections of pus may occur. Here the pus does not track back into the bowel; it gets between the two sphincters and fills the whole ischio-rectal space. At last it may, often does, as in this instance, burst externally, which means that extensive suppuration has gone on without relief until the abscess bursts. These people come to one more often for the relief of tension caused by pressure of the pus than do those with a submucous abscess. An abscess which forms in the way I have described may be either opened by the medical man or may open itself. The ultimate result in all these cases is that a fistulous track is left. So remember, when anyone comes to you with ischio-rectal abscess, not to say—"You will be well in two or three days," because the patient will not; usually they do not get well until something further has been done for them in the direction of curing the fistula, and in my experience there are very few exceptions to that. Now here, instead of having an abscess cavity discharging in the ischio-rectal region, the submucous region, or the pelvi-rectal region, what you have is a track

where the abscess was, consisting of chronically inflamed granulation-tissue which discharges pus. If this track becomes in any way blocked up you may get some accumulation of pus, but if not, these patients may go on for years without seeking relief. Last year, in August, I had a gardener sent to me at St. Mark's Hospital, who had had a fistula for sixteen years, with the result that the chronic inflammatory changes had gone on round this lesion all those years, and had so altered the musculature of the part that they were not muscles at all—they were simply masses of fibrous tissue. He had no sphincter power, and it felt like a chronic malignant growth. I could do nothing for him except a colostomy, as he had no control, and this he refused, and there was no justification for operating on him locally, because I might have made his loss of control in that region even worse than it was then. Yet he had gone on doing his work as a gardener with this chronic fistula. Of course that is very unusual and it is an extreme case, and probably a man of that kind did not care very much what his condition was. But I tell you of his case because it illustrates that these people can go on for a long time with a chronic suppurating sinus, simply because it does not occasion very much pain. And remember discomfort is largely an individual question, and some people in outdoor life do not pay very much attention to it. The danger in not doing that is that in a condition such as ischio-rectal or complete fistula you may get diverticula going all over the place. In one extraordinary subcutaneous fistula which I saw some years ago the skin had been invaded over the whole region of the buttock, and the patient had only little islands of skin and twenty-two external openings over his buttocks, and the whole skin looked like a piece of rotten leather. It had lost its elasticity; it was nothing but epidermis infiltrated with fibrous tissue.

The ordinary symptoms of a chronic fistula are that a man who has it cannot sit down very comfortably, and there is the discomfort of the discharge. With the submucous condition there is often a chronic discharge of pus and also at times of blood, and there is in addition some pain on defæcation. In the pelvi-rectal fistula the preliminary abscess condition is, from the start, grave for the patient, and during convalescence nothing must be taken for granted, but there must be absolute knowledge of the progress. In the typical fistula, such as you saw, there is an external opening, the track, and an internal opening. The original area of infection in all these cases is just within the anal margin, at the ano-rectal junction. You must always feel there and look for the original infecting lesion, and if you put a probe in at the external opening and gradually work it along the indurated tract which you feel, and put the speculum into the anus and separate it, and have a small lamp if you like, you will see the probe point through an attenuated piece of mucous membrane, which is obviously abraded, and there you will see the outlet which the micro-organisms have taken. The way to deal with this fistula is, in this instance, to pass a probe-pointed director in from the external opening and work it along the track into the opening, and then pull out the end of the director on to the buttock and divide the tissues lying above it. But there is a point in that which you should remember. If you divide the fibres between these two openings you may divide each muscle-fibre at a different angle, and instead of these joining end-to-end with a thin line of fibrous tissue, there will be a gradually increasing gap from one piece of muscle to the other, with the result that there will be an extremely attenuated sphincter, and some permanent weakness in consequence. This is a most serious thing, and to be avoided. Enlarge the *external* (skin) opening until you get it opposite the internal opening, and then pass your probe in opposite the other; then divide the tissues straight across, and they will separate in an even line. Then as they heal up they come together fairly uniformly, and there is one thin line uniting the whole sphincter muscle there. In the case of this man he happens to have very bad piles, which he has had now fourteen years. He says they bleed freely and come down when his bowels act. So what I shall do with him is to remove his piles by excision, and having removed them, the mucous membrane will be brought down to the skin. If I do the operation which I have mentioned on his fistula we shall have the mucous membrane free, and one can open the fistula right away, and starting from the inside here clear it out and get the tissues healthy and sew it up to the external opening, and bring the mucous membrane down and tack it down on to the skin at the internal opening of the fistula,

and sew it round. It is a line of treatment which I do not approve of in most cases of fistula. But here, where the man is otherwise healthy and has a chronic fistula and very bad hæmorrhoids, there is a good opportunity of doing it. Now, one or two other words about this condition, because it is one you will not see much of in your hospital work, and you should know something about it in general practice. The ischio-rectal abscess and ischio-rectal complete fistula is a, comparatively speaking, simple matter to deal with. But the submucous abscess and fistula is not simple. To deal with this a proper investigation must first be made, and if necessary the patient is put under an anæsthetic, and if this is done any operation necessary can be done at once. The best method here is to take a hook probe and pass it into the opening, and hook it down until you feel the probe just underneath the skin on the buttock outside the sphincter. Make a small incision through the skin and pull the end of the probe through; withdraw that and put a fistula-director into the opening, bringing the end out of the anus through the internal opening, and divide the overlying tissues as in an ordinary ischio-rectal fistula. Do not trust to luck and merely divide the mucous membrane inside; this may cause all sorts of trouble and is a fertile source of rectal ulceration.

With regard to the grave conditions of pelvi-rectal fistula, they may be quite serious. They often burst back higher up into the bowel. But this secondary opening should never be interfered with. Always remember that the original internal opening or site of infection is at the ano-rectal junction, and the higher opening is a secondary matter, and recognising it as such you will leave it alone. Do not put your fistula director into it and divide all the tissues lying above it. Bad results occur from such treatment, *e.g.* permanent incontinence, stenosis, and so on. The way is to work from the original site of infection, and then do one of two things. If you have to divide a large portion of the levator ani muscle and internal sphincter and external sphincter it may result in a certain amount of incontinence. Avoid that, if possible, in this way: get your original internal opening at the ano-rectal area, put a probe in it, put something else bigger into it, put a pair of sinus forceps in it, dilate it sufficiently to be able to get your finger into it, and feel all round for the cavity. By that means you will have made a very efficient drain. You will have stretched the tissues sufficiently not to let them contract down again, and so prevent a pent-up collection of pus. You can then scrape and pack this area. Very often this will produce a cure right away. It is a serious condition, and if you do too much there is a chronic distressing condition for the rest of the patient's life, and if you do not do it sufficiently it may track anywhere. What was long a favourite treatment in this condition, and is still practised by some, was to inject the sinus with silver nitrate. I have seen it done many times, and it is certainly effective in certain conditions. But I have also seen shocking results ensue from it. One of my former house-surgeons at St. Mark's had a case of fistula there, which was not going on according to his expectations, and so he thought he would treat it with nitrate of silver injection. The result was that the whole of the patient's buttocks suppurated. Fortunately the patient lived all right, and went out with a very fair amount of rectal power and control. But the risk is too great in my opinion. The reason is that when silver nitrate is injected a violent reaction is set up. It is all right for a simple straight tract, but there may be a side tract which has not been noticed, and the silver nitrate may block this up completely. Infective material is there, which is penned up and goes on increasing. There is very likely to be a large area of suppuration going on all round. But if you know your limit in the use of this remedy you can pass a probe coated with nitrate of silver down the track, with much benefit.

In these cases I use cataphoresis, with zinc sulphate, which we have instituted here, with good results. A patient who had been in Colombo for a considerable time was sent to me with a very bad fistula, and, like so many men who have been in a hot climate for a long time, he was much run down in general health, and his tissues were poor. He had had this chronic rectal trouble for some time, and although I operated upon his fistula and sent him to the seaside he still did not get well; he had the kind of persistent sinus which I have spoken to you about. As the sinus would not heal we tried cataphoresis. When we started to do this he had a track $4\frac{1}{2}$ to 5 in. long, and in seven days from the commencement of that

treatment the track was only 1½ in. long. And to-day he is practically well, yet he has only had cataphoresis twice. You must not forget this treatment for bad cases of fistula and other rectal troubles such as I have spoken of. Another thing which I should like to mention about fistula is that sometimes they are worse than they look, and you should be very careful in making any prognosis. Do not be in a hurry to say this is only a little thing and it will soon get well unless your knowledge is sufficient for you to say so with confidence.

Often in cases of pelvi-rectal fistula the track goes a long way, and may even track round and invade the bladder. In other cases the fistula may result in inflammation of the peritoneum, and from the peritoneum the inflammation may spread on to some adjacent coils in the small intestine. The small intestine may even be adherent to the inflamed peritoneum in the pelvic area.

You may find that the patient has a tuberculous fistula. I have not touched on this branch of the subject, but the area for these tuberculous troubles is the same as for the conditions I have been describing. A tuberculous infection may riddle the whole of the tissues in the neighbourhood. There may be necrosis of bone in the sacrum or the coccyx; there may be also a large opening high up in the bowel, and from it may come fæcal matter through the fistulous tract and exuding through the external opening. Those conditions are rather rare and most distressing. And when a fistula opens into the bladder flatus passes *per urethram* and cystitis occurs. In such cases there is only one thing to do. When you find there is no local operation which you can devise which will satisfactorily deal with the fistula, do a colotomy. It may be only a temporary affair, but on the other hand it may be permanent, and it is as well to prepare the patient for such an eventuality. You, by this means, remove all this area from fæcal contamination, and you can wash it out and deal with the sinuses. That gives the patient the best chance, and it often yields extraordinarily good results. I have operated upon a man who was almost in a dying condition from septic absorption from a multitude of fistulous tracts, opening into the bladder and elsewhere, with a high temperature and rapid pulse due to septic absorption. I did a colotomy on him, and the result was that at the end of three months his sinuses healed up. If the rectal mucous membrane is not too much destroyed he may be in a position to have the colotomy closed.

I have to-day run over most of the main points with regard to fistula and other similar affections of this region which can be easily cured by anybody. But remember it is rash to make a prognosis unless you are certain you know the possibilities and the exact condition of affairs. If there are points in the cases about which you are uncertain, give a guarded forecast. There is always the possibility that these cases may go on for weeks or months, and unless you have confidence in yourself, and, above all, unless your patient has confidence in you, you may have a considerable amount of discredit because of something which has happened and which is not your fault. With regard to the dressing, chronic cases will heal up to a certain point, and then you may find there is something which you have not previously noticed. You examine again and find that some pus is welling up from some tiny opening in the granulation tissue. On putting in a probe you may find a deep-lying cavity which you have to open up again. That condition is called bridging. You must expose the whole of this infective cavity and pack it and let it heal up again from the bottom. This bridging occurs in cases which are not properly dressed and carefully looked after by some properly instructed nurse, or by a practitioner who knows what he may reasonably expect, and be on the look-out for, in chronic fistulæ.

September 19th, 1910.

Technique of the Injection of the Ehrlich-Hata Remedy.—Wechselmann and Lange appear to have arrived independently of and simultaneously with L. Michaelis at the determination to inject the new preparation in a neutral suspension. They rejected phenol-phthalein as an indicator, however, believing it to be destroyed in the reaction. Their original solution of the remedy is made with soda lye. Glacial acetic acid is used for neutralising, and ordinary litmus paper as an indicator. Methyl alcohol should not be used as a solvent, for it alone can cause amaurosis in the supersensitive, and this might be set down to the remedy itself. The authors recommend as an injection site the region beneath the shoulder blade.—*Medical Record*, vol. lxxviii, No. 9.

THE EARLY DIAGNOSIS OF SUBACUTE COMBINED DEGENERATION OF THE SPINAL CORD.*

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GENTLEMEN,—Subacute combined degeneration of the cord is a comparatively new disease, and, like most diseases which have not long occupied positions of their own, is undergoing a gradual process of illumination and disclosure. Our first distinct conception of the disease, for which we are chiefly indebted to a paper written by Russell, Batten, and Collier about ten years ago, has been, and is being, considerably modified by further experience, and, if we are to profit by the fruits of this experience, a short time devoted to the more important additions to our knowledge will not be wasted.

Among those nervous diseases with which any medical man may reasonably expect to be confronted there are few more difficult to diagnose in its earliest stage, and few, therefore, more dangerous to a reputation for reliable powers of prognosis. My remarks to-day will be directed to this important aspect of the disease—its early recognition.

Subacute combined degeneration of the cord is one of the clumsiest titles in medical nomenclature, but it will probably be retained for two reasons: In the first place no one has been able to suggest a better; in the second place it possesses the advantage of conveying, clearly if not very concisely, the essential pathological features of the disease. Without going into details of morbid anatomy, I would remind you that a degenerative process mainly affecting the long afferent and efferent tracts of the spinal cord constitutes what is found post-mortem in fatal cases, and is responsible for their clinical charcteristics during life. You may ascertain from any text-book that it is a disease of middle life, of unknown ætiology, often, but not always, associated with grave anæmia; that its onset is insidious, and its course progressive, remissions being the exception rather than the rule, and that a fatal termination is common any time from six months to three years after the appearance of the first symptoms. Further, you will learn that the first stage, roughly that period intervening between the onset and the date when the patient loses his power of walking, is characterised by (*a*) paræsthesiæ, such as numbness, tingling or burning in both hands or both feet, or simultaneously in all four extremities; (*b*) some paresis and impairment of sense of position in the feet and hands, evidence of which is afforded by the slightly ataxic gait and rather clumsy hands; (*c*) loss of abdominal reflexes, exaggerated tendon-jerks, ankle clonus, and extensor plantar responses.

This is, briefly, the generally accepted description of the first stage of the disease, and I propose to demonstrate to you by means of actual cases that this description may be very misleading unless it is admitted to apply only to the majority. There is little fault to find with (*a*) and (*b*), but when it comes to (*c*) we shall find that in certain instances very different results may be obtained from the examination of the reflexes.

CASE I.—Here is a man, æt. 43 years, who has never had any venereal disease, and who, until the end of 1908, always enjoyed perfectly good health.

In January, 1909, while feeling rather out of sorts, he noticed some numbness of the left leg and some weakness of the left leg and left hand. Slight at first, these symptoms became gradually more marked, until in August he was obliged to give up his work. Weakness of the right hand followed, and the numbness on the left side became associated with a "sore feeling," especially noticeable if he rubbed himself on the side of the trunk or on the arm or leg. He was admitted into the hospital on May 11th, and on May 27th he developed some numbness and burning in the right foot.

You will notice that he is well nourished, and, although of sallow complexion, not obviously anæmic. Walking in the ordinary way, there is little to attract attention in his gait, but if I ask him to walk along a straight line, placing one foot immediately in front of the other, he displays a decided amount of ataxy, which is increased if he attempts to take his eyes away from his feet. Further examination shows that all his cranial nerves are carrying out their function in a normal manner. The left arm is weaker than the right, and the strength of the latter is by no means up to

* Lecture given at the National Hospital for the Paralysed and Epileptic.

the mark. Testing his sense of position by getting him to close his eyes and bring his forefinger to the tip of his nose, it is clear that there is some defect, at any rate on the left side. The trunk muscles are fairly strong, although he is unable to raise himself to a sitting posture without the help of his hands. The superficial reflexes of the abdomen are entirely absent. The lower extremities are slightly spastic, and some paresis can be demonstrated when he is asked to dorsiflex the ankles or flex the knees against any resistance. The spasticity and paresis are more marked in the left than in the right leg. The knee-jerks are very brisk; ankle clonus and extensor plantar responses can be obtained on either side. With the exception of the paræsthesiæ and the slight but definite impairment of sense of position, disturbances of sensibility are almost, if not altogether, wanting. Perhaps the prick of a pin is not quite so painful on the legs and abdomen as it is on the upper part of the thorax, but it is difficult to be sure that there is any real defect. The control over the sphincters is perfect, although now and then micturition may be a trifle hesitating.

The history of this man's illness and the clinical picture he now presents correspond closely to the classical type of the first stage of subacute combined degeneration of the cord except in one detail, namely, the lack of symmetry in the evolution of his symptoms. From the beginning the left side has been more affected than the right and to a degree which is more marked than I have yet observed in cases of this disease. When I first saw the patient in the out-patient room the asymmetry was more striking than it is at the present time and one of my objects in showing him to you to-day is to impress on your memory the possibility of such asymmetry occurring.

For the purpose of contrast let me now show you another patient.

Case 2.—This patient is a man, æt. 45 years, and was admitted into the Royal Free Hospital under the care of my colleague, Dr. Carr, on December 24th, 1909, on account of abdominal pain and vomiting which had persisted over a period of nine weeks. He was thin and cachectic, but, with the exception of some pyorrhœa alveolaris and slight abdominal tenderness, presented no definite physical signs. His gastric symptoms rapidly cleared up, and he left the hospital practically well on January 6th, 1910. Within a few days of his discharge he noticed some numbness and weakness of his feet and numbness and tingling in his hands. These symptoms persisted and he was re-admitted on April 6th, when he was found to be anæmic, although better nourished than he had been three months previously. On examination he was found to have lost his knee- and ankle-jerks. Cutaneous sensibility appeared to be perfect and his plantar reflexes were of the flexor type. Three weeks stay in hospital produced no improvement, and when I examined him on May 7th he presented much the same signs as he does at the present time. Meanwhile, the numbness and tingling had spread up his legs and on to his forearms.

You will notice his marked pallor and his well-nourished bodily condition. In attempting to walk along a straight line he displays just the same amount of ataxy in his gait as did Case 1. There is nothing wrong with his cranial nerves. His grasps are about equal but not up to the normal standard. Carrying out the finger-nose test with closed eyes he exhibits a slight degree of inaccuracy but no tremor. The strength of the trunk and leg muscles is not what it should be, although he can carry out all movements with fair power. In particular there is decided paresis in dorsiflexing his ankles against any resistance. No loss of cutaneous sensibility can be detected with certainty, but I must draw your attention to the fact that, although a month ago his leg muscles were more than usually sensitive to deep pressure, at the present moment they are, if anything, hypo-algæsic. Moreover, he is not always accurate in discriminating between the head and point of a pin applied to his lower extremities.

So far the physical examination of this patient presents results which differ little from those which I demonstrated in the last case. When we come to test his reflexes, however, there is a marked contrast. It is true that the abdominal reflexes are absent in both cases, but in this patient the tendon jerks, instead of being exaggerated, are entirely wanting, ankle clonus is not obtained, and the plantar responses are quite indefinite in character. With regard to the latter I must point out that they are probably in a transitional stage. A few weeks ago they were definitely flexor; now the response is unreliable, sometimes inclining to extension and sometimes to flexion.

If we analyse the results of our examination I think you will agree that there is evidence of some affection of both the efferent and afferent tracts of the cord. On the one hand there is the paresis and the absence of abdominal reflexes pointing to interference with the normal function of the pyramidal tracts; on the other hand the ataxy, the paræsthesiæ and the absent tendon jerks suggest disease of the posterior columns and other afferent systems. There is sufficient justification, therefore, for assuming the presence of some combined degeneration of the cord. The history of the onset, associated as it was with some abdominal disturbance, and the marked anæmia lead me to form the opinion that the patient is suffering from subacute combined degeneration of the cord in its first stage.

Perhaps I should not be so willing to come to this conclusion if it were not for a recent experience, which I will relate as briefly as possible. In June, 1909, I was consulted by a gentleman, æt. 60 years, who had been in good health until the preceding March, when he complained of burning and numbness in his hands. Some weeks later he developed a girdle sensation and then burning and numbness in his lower extremities. When I examined him he was sallow but not anæmic. He described his legs as feeling numb and cold and his feet as if he was treading on something hot. There was some hesitation in micturition but no loss of sphincter control. There was no loss of power, but considerable ataxy of the legs and clumsiness of the hands due to impaired sense of position. He had a curious mixture of hyperalgæsia and hypo-algæsia on his legs and trunk, so that a pin-prick was very painful in one spot and not recognised as a point in another. Deep pain sensibility was diminished. The abdominal reflexes were present, but all tendon jerks were wanting and the plantar responses were flexor in type.

I formed the opinion that the patient had some affection of the posterior columns, but did not care to be certain that he was suffering from tabes dorsalis, because he gave a negative Wassermann reaction, because of the rapid development of symptoms, and because he had no cranial nerve troubles, and, particularly, no Argyll-Robertson pupils.

Seeing him again in October, I found the clinical picture entirely changed. The former symptoms were present in more marked degree, but he had in addition great loss of motor power and considerable rigidity of the muscles of the trunk and all four extremities. It was now possible to obtain the knee-jerks, and the plantar responses were more extensor in type. He had, in fact, developed all the signs of disease involving the lateral columns in addition to those denoting posterior column degeneration. I did not see the patient again, but I ascertained that he went rapidly downhill and died at the end of December.

These are not the only cases in which I have met with diminished or abolished tendon-jerks in the first stage, and accumulated experience leads me to believe that the condition of the reflexes is an unreliable guide to the diagnosis of early subacute combined degeneration of the cord. We shall make fewer mistakes if we remember not only that both the posterior and lateral columns of the cord are affected in this disease, but that the affection of either the posterior or of the lateral columns may precede and preponderate over the other. Early involvement of the posterior columns will give a picture in which afferent symptoms and absent tendon-jerks constitute the more prominent features; on the other hand, excessive changes in the lateral columns produce a condition in which spastic paraplegia with exaggerated jerks and extensor plantar responses are the more striking phenomena.

As an example of the latter type of case I will draw your attention to some sections of a spinal cord which was given to me by Dr. C. R. Box, and which was taken from a case of subacute combined degeneration of rather unusual character. In the first place, the patient lived for six years after the initial symptoms of the disease made their appearance; in the second place, you will see that the degeneration in the lateral columns far exceeds that which is present in the posterior columns. As a general rule death takes place at an earlier period, and the affection of the lateral and posterior columns in the final stages is more or less equal in degree. Judging from this as well as from other cases, I am inclined to believe that the prognosis with regard to duration of life is somewhat better in those patients who present predominating lateral column signs than in those in whom the posterior columns appear to be more severely affected. Some corroboration of this opinion may perhaps be derived from the fact that in cases of this disease the appearance of marked sensory changes, associated with loss of tendon reflexes, is the recognised signal for the near approach of a fatal termination.

Having laid emphasis on the state of the reflexes

in the first stage, I must not omit to refer to one or two other points of importance in the diagnosis of the disease. With regard to anæmia, it can only be said that this is a very variable quantity; it may be a prominent symptom or it may be entirely wanting. In not a few instances it appears, but yields rapidly to treatment by arsenic, perhaps to re-appear again after an interval. The blood-changes are not specific and only rarely conform to the characteristics of pernicious anæmia.

Gastro-intestinal disturbances, associated with bouts of moderate pyrexia, are frequent, and may precede or accompany the earliest signs of disease of the nervous system. Their occurrence may sometimes be of value in forming a diagnosis when nervous symptoms have not reached any degree of severity. Vomiting or diarrhœa in these attacks is often associated with a dull aching pain in the epigastrium or right hypochondrium.

I have already referred to the increased sensitivity to superficial or deep stimuli of a painful character, which may be present before any diminution of sensibility can be detected by ordinary methods of testing, and I have on more than one occasion observed at this stage a peculiar condition of the skin, especially of the hands and feet. The palms of the hands, the soles of the feet, as well as the palmar and plantar aspects of the fingers and toes are unusually red, almost inflamed-looking; at the same time there is a tendency to superficial desquamation of the epithelium. This may be accompanied by a subjective sensation of disagreeable heat in the affected parts of the extremities. By the time that the second stage of the disease is reached these phenomena are usually much modified, if they have not altogether disappeared.

Having referred to several points which may be of service to you in recognising this disease, it is a sad necessity to add that the prognosis is always extremely grave and that our means of retarding its progress are at present inadequate. The general features of the malady strongly suggest a toxic process, having its origin in the alimentary canal, and it is not surprising to find, as I have found, that measures directed towards intestinal disinfection appear to meet with more success than any others, although even long periods of retardation or, perhaps, slight improvement end in relapses which leave the patient worse than he was before. London hospitals and nursing homes do not suit the sufferers from this disease; far better results can be obtained by removing them, when it is possible, into the country, and into conditions where they can obtain a maximum of fresh air and plenty of good food. Tonics, with the exception of arsenic, are of little value, and electricity has no effect. Massage and Fraenkel's exercises may often ameliorate the disorders of locomotion under the more favourable circumstances I have described.

September 19th, 1910.

THE DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS OF THE PATHOLOGICAL PROCESSES CAUSING ENLARGEMENT OF THE KIDNEY.*

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My remarks are based upon an experience in abdominal surgery covering a period of sixteen years, and are merely intended to represent the writer's personal views and those methods that he has found most serviceable in making the diagnosis of renal neoplasms and other lesions causing enlargement of the kidney.

As in every other branch of surgery, affections of the kidney require a complete history of the case in order to arrive at a correct diagnosis. Patients often make mistakes when their opinion is asked about the size and growth of the tumour present, but generally they will be able to accurately give the starting-point of the tumour, and very much depends upon this fact. In most cases there are disturbances in digestion, and after a certain lapse of time vomiting occurs and later on emaciation. The patient will also complain of headache and pain in the renal region, which in some instances is not especially marked, but it is continuous and of a rather dragging, dull kind. In other cases the pain occurs only occasionally, and is then severe and sharp, radiating toward the thighs. Disturbances in the micturition will be noticed, such as some diminution in amount, or, on the other hand, there may be polyuria. Blood or pus may be found in the urine. These symptoms, when combined with a swelling in the abdomen, are usually enough to cause the patient to consult the surgeon.

On inspection, when the tumour has reached a certain size, swelling of the abdomen will be readily seen when the patient lies on the back or side. It may also project into the lumbar region, but one should not conclude from this fact that a renal enlargement exists. Extensive dilatation of the abdominal veins is of little consequence, as far as the diagnosis is concerned, because they are met

* Delivered before the American Urological Association.

with in many other affections, particularly in hepatic cirrhosis.

Palpation is far more important than inspection, and numerous are the methods that have been advised. Guyon has insisted particularly on renal *ballottement*. Much may also be obtained by feeling the border of the liver and spleen, but in point of fact this is frequently a difficult matter, particularly when adhesions are present. The consistency of the tumour should be noted, and if a grating noise may be elicited it is suggestive of renal calculi. Palpation of the growth is possible when the patient is not too fat, and when the tension of the abdominal walls is not too great. The lower pole of the tumour can usually be made out, because it does not usually sink behind the iliac crest. Its posterior internal border may sometimes be rendered difficult of detection when a marked physiological lordosis exists. The intestine should always be thoroughly emptied before the examination is undertaken.

Placing the patient on the normal side will sometimes be of service; one examining hand is placed on the anterior abdominal wall, the other over the lumbar region. During respiration the tips of the fingers should be made to glide over the lower pole of the kidney. Although it has been upheld that the kidney does not move with the respiration, I have, in quite a few cases where the organ was enlarged and not adherent, been able to prove that it does follow the respiration. It begins its excursion at the commencement of inspiration, and continues to the end of the act, while the liver excurses only towards the end of inspiration.

Ballottement is, perhaps, the best diagnostic proof that we have for proving that the tumour really lies in the lumbar region, provided it has not reached such a size that it has become adherent to the anterior abdominal wall. When this has occurred the renal origin of the growth can often be established if the other abdominal viscera can be palpated separately from the growth. The renal origin of a tumour can furthermore be established in many cases by its retroperitoneal position, as is shown by the presence of the ascending or descending colon running over the anterior surface of the tumour, and can be felt there as a narrow, firm band, which can be rolled under the fingers when it is empty; should it contain gas it will be detected in the form of a soft, sausage-shaped protrusion. Frequently, however, it cannot be felt, particularly on the right, where, on account of the position of the intestine in relation to the kidney, the former is frequently pushed inwardly or downward. These statements apply only to tumours which have not reached an excessive development, and are of no value in those instances where the growth occupies the entire half of the abdominal cavity.

Confusion with other tumours is quite possible when portions of the intestines are present over the anterior aspect of hepatic or ovarian growths as a result of adhesions. Thus, for example, the small intestine was found lying across the neck of a cystic gall-bladder. In these cases palpation will only be of value when the border of the liver can be distinctly made out from that of the tumour. Then, again, one cannot always exclude the hepatic origin of the growth from the mere fact that the free edge of the liver can be made out, because a neoplasm may arise in the under surface of the liver.

The nature of a renal growth is not always easy to ascertain by simple palpation. However, it may be said that there are three pathognomonic results, movable kidney being excluded, which, however, are not frequently encountered. These are—(1) hydatid crepitus; (2) a grating sound when several large stones are present in the renal pelvis in thin subjects with flaccid abdominal walls. This is a very rare occurrence, but such cases have been encountered; (3) shrinking of the tumour when pressed upon, in which case one is dealing with a retention cyst which has discharged a portion of its contents into the bladder. The presence of fluctuation is often very difficult to detect, and even when it can be the nature of the cystic growth cannot always be surmised.

I will particularly insist upon the fact that *ballottement* may be elicited in certain cystic growths arising from the under surface of the liver, and consequently this sign must never be considered as absolutely pathognomonic as far as cystic growths of the kidney are concerned. Then, again, any cystic growth of the omentum or spleen may give rise to *ballottement*, provided that the growth is in contact with the posterior abdominal wall.

A renal growth sinks down to the iliac fossa

when it extends as far as the median line, and when not very large it is never in direct contact with the anterior abdominal wall. Under these circumstances it can usually be pushed back into the lumbar fossa with ease. On the other hand, tumours lying under the liver are not separated from the latter by a tympanitic zone. They will be found to follow the respiratory movements when the patient is lying on the back, and will rise toward the costal margin and will then show a distinct hollow behind the anterior abdominal wall, and they cannot be reduced into the lumbar fossa.

Generally speaking, palpation of the kidney with the patient lying on his side, when alone resorted to, is not of any great importance, and I feel that it is the most difficult of all methods of palpation, and is only of real value when the renal enlargement is still limited in extent.

Attention should also be called to certain cases in which a certain portion of the kidney is the seat of a cystic or solid growth, and the remainder of the organ retains its shape and rests upon the tumour like a cask. When this condition of affairs can be made out it is valuable from the diagnostic standpoint, because in the case of an abdominal tumour of doubtful nature it is characteristic of the kidney alone. The relation of the ascending or descending colon to the anterior aspect of the tumour is of diagnostic value only inasmuch as it shows that the growth is retroperitoneal.

Percussion is also of value in the diagnosis of a renal tumour. When the kidney is normal, or when the patient is very stout, percussion is of no use, but it is of importance when the kidney is enlarged. The colon is pushed forward and inwardly when the growth is large, and is usually flattened out, and if its exact location is to be detected it should first be artificially distended. As a characteristic sign of a renal tumour, I believe that the palpable borders are never the same as those found by percussion. I have never been able to percuss the internal border of the enlarged kidney. The upper border of the right kidney cannot be separated from the liver dulness, but that of the left can occasionally be separated from the splenic dulness, particularly in elderly people. The external border can be mapped out in almost all cases, likewise a distinct space can always be found between the lower pole of the kidney and the iliac crest. If there is much fat and œdema in the renal region, enlargement of the liver or ascites, the result of percussion is rendered unsatisfactory.

Exploratory puncture of a renal tumour in the lumbar region has often been recommended, and in certain affections, such as hydronephrosis, echinococcus cyst, or cystic kidney, it is certainly of advantage upon the condition that operation is immediately carried out, because otherwise there is danger of infection. Puncture, which formerly was considered important for the discovery of malignant growth-cells, is of little value, and probably will fail in most instances.

The fluid removed from a hydronephrosis is usually clear, with a specific gravity of 1008 to 1020. Its composition may have undergone considerable change from secondary processes. For example, it may become cloudy and albuminous from admixture of mucus and pus. Microscopically uric acid crystals are found. In cystic kidneys there is always polyuria, and consequently the specific gravity is much diminished. The fluid may also contain altered red blood-cells and cells containing pigment of a peculiar dark-brown hue. Fluid from an echinococcus cyst is usually very clear, of neutral or alkaline reaction, a low specific gravity, rich in sodium chloride, which crystallises on evaporation. Albumen is absent, or if present, only a trace can be found. Succinic acid is said to be a constant constituent.

Referring now to exploratory incision, it is my opinion that when this is done for diagnostic purposes laparotomy should be resorted to, because it gives a view of the entire abdominal cavity, and what is still more important, the supposedly normal kidney can be palpated. At any rate, the operation of the diseased kidney should immediately follow the exploratory incision so as not to submit the patient to two interferences.

To sum up the question of physical examination, I would say that inspection, percussion of the lumbar region, as well as an exploratory puncture, are of no particular value when taken alone. The results are of greater value by percussion over the anterior aspect of the growth, while the most important of all is palpation. The lateral position of the patient is not, to my mind, nearly as good as the dorsal position, but in some cases both may be resorted to with advantage. The cases where

exploratory laparotomy is indicated are—(1) those in which the diagnosis of malignant growth is uncertain; (2) in certain cases of anuria; (3) to ascertain the presence and condition of the other kidney; and (4) in severe contusion of the kidney.

The use of the cystoscope and ureter catheter must also be resorted to, as much valuable information may be derived therefrom. The condition of the urine flowing from each ureteral orifice, likewise the manner in which it is projected, are ascertained. The ureteral catheter is of use in detecting obstruction of the ureter and also the presence of a small hydronephrosis, but when the latter lesion has caused a large dilatation of the renal pelvis this mode of investigation will usually be found of no use, because some abnormal condition of the ureter has occurred which will prevent the instrument from reaching the kidney. Personally I have been well satisfied with the use of Luys' separator in order to obtain the urine from each kidney, and having had a very large experience with this intrument, I feel that it may be safely advocated provided that the technique of its employment, as exposed by its originator, be strictly followed.

Considering now the question of renal tumours, it may be said that the diagnosis can be made by palpation, the changes in the urine, and hæmaturia. The cystoscope will show blood coming from the uterer of the diseased side if the kidney happens to be bleeding at the time. In some cases cloudy urine will be seen coming from the ureter.

Catheterism of the ureter will give the same results as the cystoscope, namely, blood or cloudy urine, in which one may occasionally be fortunate enough to find fragments of the growth. In cases of so-called essential hæmorrhage palpation will remain negative, and hæmaturia is the only symptom present. The cystoscope will show blood coming from the ureter while catheterism will reveal nothing more. Aneurysm and hæmatic cysts of the kidney, which are extremely rare, cannot usually be demonstrated by palpation, and they will give rise to the presence of blood in the urine only when they have ruptured into the renal pelvis.

Echinococcus of the kidney cannot be diagnosed when the sac is very small, but when it has attained some size it may be detected by palpation. The cystoscope and ureter catheterism are valueless. Pyonephrosis, whether due to tuberculosis or some other infection may give rise to enlargement of the kidney, but in these cases the patient is weak; there are fever, pain, and marked changes in the urine.

The diagnosis of hydronephrosis is made by palpation; the changes found in the urine, polyuria, oliguria, anuria, etc. The cystoscope and ureteral catheterism will make it evident that no urine flows from one of the ureters. In cases of small hydronephrosis the urine may be drawn by the ureteral catheter, after which the size of the tumour will have diminished. But this only applies to moderate dilatation of the renal pelvis, and in the large hydronephrosis there is almost invariably an obstruction of the ureter which cannot be overcome by the catheter.

Large hydronephrotic tumours conceal their evolution behind countless syndromes. They have often been mistaken for ovarian cystomata; their lower pole extending into the small pelvis could easily be felt *per vaginam* in the form of a fluctuating mass on one side of, or behind, the uterus, and quite independent of the latter. I am aware of ten instances where this condition occurred.

In other instances the tumour took on the aspect of a hydatid cyst of the liver or of the pancreas, and in one of my cases a calculous hydronephrosis was accompanied by icterus from compression, thus simulating a pancreatic growth.

In these cases this renal lesion has simulated appendicitis, with violent paroxysms of pain, vomiting, and other digestive disturbances. The tumour extended into the right iliac fossa and was mistaken for an appendicular abscess. Operation revealed the true condition and a healthy appendix.

In tuberculous subjects a hydronephrosis is often overlooked, and is only found at autopsy. I have met with one such case in which the only symptoms complained of were indefinite renal pain and severe pain on micturition.

A hydronephrosis may also closely simulate renal lithiasis, pregnancy, or a lesion of the pylorus.

There is no diagnostic criterion, but ureteral catheterism is useful and should never be neglected, for often it will affirm the diagnosis and reveal the physiological condition of the other kidney. The areas of dulness and tympany should be mapped out by percussion, which will at least indicate the retro-peritoneal location of the tumour, and the

examination should always be concluded by a careful analysis of the urine from each kidney when it can be obtained.

As to the urine from a diagnostic standpoint, it may be said that traces of albumen can be demonstrated in almost every renal affection. It is found in nephritis and cystic kidney. Hæmaturia is encountered in carcinoma and sarcoma. Blood-pigment crystals, fatty granules and epithelial elements are also found. It must be said, however, that in some cases of renal tumour, even when far advanced, no changes in the urine can be detected.

The large polycystic kidney is ordinarily bilateral. The kidneys may reach enormous dimensions, even extending as far as the pelvis, and may occupy the greater part of the abdominal cavity, and it is in just these cases that a differential diagnosis between ovarian cysts must be made.

A polycystic kidney is composed of a number of cysts of various sizes, the largest being the size of a small apple, while microscopical ones are also present. The contents of the cyst resemble bile and some are a brownish red, this being due to hæmorrhage.

In many cases the patient presents the typical symptoms of chronic nephritis, and unless the renal region is palpated no enlargement of the kidney will be evident. In these cases there is polyuria; the urine is of low specific gravity and contains albumen; there is cardiac hypertrophy and anæmia. Hæmaturia occurs, and in this it is distinctive from the small contracted kidney. The urine contains a large number of casts, both granular and hyaline.

Palpation reveals an absolutely characteristic condition of affairs, the kidney being felt much enlarged, hard, and with an irregular surface. This affection is differentiated from malignant renal growths by its very chronic progress, which lasts for years, and because it usually involves both kidneys. The simultaneous presence of cysts of the liver, which can be easily palpated, helps the diagnosis. Lejars collected sixty-three cases of polycystic kidney, and in eleven instances there were multiple cysts in the liver. It is probable that this affection is congenital, and only develops as times goes on.

The differential diagnosis must be made between hydronephrosis, echinococcus, and ovarian tumours. These, contrary to the polycystic kidney, are usually unilateral and fluctuate. The polycystic kidney is very hard, and fluctuation can rarely, if ever, be detected.

Renal tumours always have the colon in front of them, while ovarian tumours hardly ever present this condition.

The symptoms of echinococcus cyst are not characteristic in the beginning, and not until the cyst has reached quite good-sized dimensions can a tumour be felt, and it is then hard and can only be made to fluctuate if a portion of the fluid is removed by puncture. Naturally the most important diagnostic signs are to be sought for in the microscopic and chemical examination of the fluid.

Large hydronephroses develop gradually and slowly displace the ureter. This lesion is probably much more frequent in women than in the male, and therefore confusion arises in the diagnosis between a renal tumour and ovarian cystoma. Nevertheless, the diagnosis can usually be made. In the first place the location of the tumour, as long as it remains small, is in the lumbar region, and then, again, one can palpate the lower pole of a large hydronephrosis above the symphysis. And lastly, the position of the uterus will establish the diagnosis. The colon lies in front of the renal growth, whether the latter be large or small, and if the lower pole of the tumour be seized by the hand it can usually be forced up to a certain extent into the lumbar region. The hydronephrosis can then be palpated in its anatomical position. If, however, the tumour is adherent, or only slightly movable, air may be injected into the rectum, and the course of the colon over the anterior aspect of the tumour can then be made out.

The differential diagnosis between ovarian growths and cystic enlargement of the kidneys can ordinarily be made with ease, but, nevertheless, an erroneous diagnosis may be made, even by those most skilled, and the mistake is not discovered until the abdomen has been opened. Both hard and cystic renal growths may be mistaken for ovarian tumours. Solid tumours of the kidney, whether benign or malignant, may closely simulate a malignant pseudo-colloid or cysto-sarcomatous growths, while various types of ovarian cystomata may assume an appearance very like pyelitis, pyonephrosis, cystic transformation, and echinococcus of the kidney. Although occasionally coils of intestine are found in front of ovarian growths and behind movable renal tumours, this is very exceptional. Large tumours of the right kidney usually have the ascending colon running along their inner aspect. When the gut is found running over the anterior surface of an abdominal

growth, this is a signal for a careful examination of the urine. Nevertheless the urine may be found normal, because the healthy kidney alone is secreting. If there is any doubt as to whether or not the colon can be felt, inflation of the gut should be resorted to, and then both percussion and auscultation will immediately settle matters. Ovarian cystomata and renal cysts may both change in size. When the kidney is the seat of the disease it may discharge its contents into the bladder, while an ovarian cyst can only discharge into the bladder when it is adherent to this organ and has ulcerated into it. Dermoids are more prone to do this than any other type of ovarian growth, so that the cystoscope and analysis of the urine will usually clear up the diagnosis. If an exact history of the case can be obtained, and one can be assured that the growth was first noticed between the costal border and the iliac crest, it may be assumed that it is renal in origin. If the growth is from the ovary the patient will have first noticed it in the hypochondriac region, and then it will have developed upwards. Ovarian cysts, when very small and with a long pedicle, can alone be mistaken for an enlarged movable kidney. In tumours of the kidney we will get the history of hæmaturia, renal colic, or considerable differences in the amount and aspect of the urine, while in ovarian tumours, unless there are pressure symptoms on the bladder, nothing will be noted as far as urinary symptoms are concerned. If all these facts are considered a diagnosis can be made in the majority of cases.

Retroperitoneal lipomata may give rise to some difficulty in the diagnosis, because, as they fluctuate, they may be mistaken for a cystic tumour of the kidney. Symptomatically they only cause discomfort. Cysts of the pancreas and spleen must also be recalled, but generally their diagnosis will not be difficult.

If a cystic tumour has been discovered, the age and the history of the patient will usually allow one to exclude the diagnosis of a malignant growth, likewise a pus kidney, when there is no elevation of the temperature and no bladder symptoms, but echinococcus and hydronephrosis may be a matter of considerable diagnostic difficulty. I would repeat again that no fluctuation can be elicited in polycystic kidney.

We now come to the consideration of malignant renal growths. Sarcomata are more frequently encountered in childhood; they are large mixed neoplasms (adeno-sarcoma, rhabdo-sarcoma); they break through their capsule at a late date in the process, but before this takes place they frequently set up inflammatory adhesions. Carcinoma are felt as large growths, break through the capsule at an early date, and when seen are usually immovable. Otherwise, the symptoms between the two types of neoplasm are the same. Sarcoma of the kidney may occur at any age, but it is most frequently met with between the first and fifth years of life and after fifty years of age. It grows quite rapidly, its surface is rather irregular, and in consistency it may be either soft or hard. The colon and duodenum are often involved in the process, and the result of this is dilatation of the stomach. Œdema of the lower limbs occurs as a result of compression of the inferior vena cava. When they have reached a considerable size the diaphragm is pushed up, so that dyspnœa results.

A very important symptom, when present, is a left-sided varicocele; this has not been infrequently met with in adults, but it must be very rare in children, as I can only find it recorded in two cases—once by Czerny and once by Bastianelli.

The diagnosis of a malignant renal neoplasm can be made with relative ease if hæmaturia and a tumour are both present. If palpation reveals an enlargement of the kidney which is not very painful, one has very good reason to suspect the nature of the growth. By exclusion and the use of the cystoscope one will be able to eliminate papilloma or epithelioma of the bladder. Renal lithiasis gives rise to severe spontaneous pain, likewise pain on palpation, while the kidney is not increased in size unless a uronephrosis has taken place from obstruction of the ureter by a calculus.

In malignant disease the migration of vermiform clots can cause pain, but it is nothing like that resulting from the passage of a calculus. In calculosis there is nearly always a certain amount of pyelonephritis, so that the urine is cloudy, while in renal cancer the urine becomes clear after the bleeding has ceased.

Papillomata or epitheliomata of the renal pelvis also bleed, and the kidney may be increased in size, but only intermittently when the ureter becomes clogged by blood-clot.

A large kidney which does not vary in size, or only slightly so, is generally the seat of malignant change, and any sudden increase in size is due to a hæmorrhage into the tumour substance or renal pelvis. This bleeding may so exactly simulate renal colic that this fact should always be recalled.

The hæmorrhagic type of renal tuberculosis is characterised by hæmaturia, and in some cases the writer has seen it reach such intensity that it completely overshadowed all other symptoms, so that malignant disease of the kidney or a hæmaturic nephritis was suspected, but the kidney was not sufficiently enlarged to be palpated, so that malignant growth could be eliminated.

A very rare form of renal tuberculosis is the so-called neoplastic type, where a tumour in one of the lumbar regions is the first symptom attracting attention, the true nature of which is often most difficult to determine.

The principal symptoms of malignant disease of the kidney are, pain in the hypochondrium of a drawing nature, and when the tumour becomes large

there is a feeling of pressure and intra-abdominal distension. The growth frequently develops rapidly, is usually rounded and of soft consistency, and often it is bound down by adhesions. Hæmaturia does not always take place, but it will be encountered at some time, usually towards the terminal period of the affection. When it does occur it is of the intermittent type, and may or may not be accompanied by pain. When the kidney is not bleeding the analysis of the urine is negative. Other symptoms arise, which are due to the presence of adhesions. When the disease has advanced there is loss of appetite, vomiting, local œdema and cachexia. Nevertheless, there are some cases where the progress of the affection is unattended by any symptoms.

The clinical picture may be divided into two symptomatic groups, which, when they occur together, make the diagnosis easy, but in the beginning of the process they are not always easy to interpret. These two groups represent the functional disturbances and the physical signs. To the former belong hæmaturia, pain, and cachexia; the latter are dependent upon the growth of the tumour. In the adult, hæmaturia occurs in more than one half of the cases of sarcoma, while in carcinoma it is present in 75 per cent., and of these it occurs in 26 per cent. as the first and initial symptom.

The character of the hæmorrhage indicates a renal neoplasm, when it occurs repeatedly, without any history of trauma, and is continuous for some time and in fairly large amounts.

Local pain is very rarely absent thoughout the entire course of the disease, but in children it is very apt to be wanting, in spite of the frequently very rapid development of the tumour. As an early symptom of the disease in the adult it occurs in about 28 per cent., while in children in only about 7 per cent. (Chevalier).

Loss of weight and cachexia, as the only early symptoms of the disease, are, according to Chevalier, noted in the adult in 18 per cent., and in children 19 per cent. The most constant sign of malignant growth is tumour, which is absent in only a very small number of cases.

For diagnostic purposes malignant renal growths may be divided into three groups. To the first belong those cases in which by palpation a growth has been found with absence of every other symptom. If only a tumour can be found, errors are frequently committed as to its mobility and size, for the former for the reason that the viscera which are adherent to the tumour are very movable, and for the latter because the development of malignant renal growths generally occurs in an upward direction into the subdiaphragmatic space, where it is difficult to palpate them. The reason for this is that frequently the growth develops in the upper pole of the kidney. This is especially true of the left kidney because there is no large organ on that side, and tumours of considerable size may escape recognition, so that a negative result from palpation does not signify that there is no tumour present.

In the second group of tumours we have those cases where the kidney is enlarged and hæmaturia is also present. It would seem from this that the diagnosis in these cases would be an easy matter, but frequently this condition of affairs simulates nephrolithiasis, and often even with the use of the ureteral catheter and the X ray there is still doubt, so that an exploratory incision must be resorted to.

In the third group may be placed those cases where there is hæmaturia without a palpable tumour. A differentiation between renal hæmorrhage and bladder hæmorrhage is readily cleared up by the use of the cystoscope. However, it is important to bear in mind that in some cases the involved kidney does not give rise to bleeding, and the hæmorrhage observed comes from the otherwise normal kidney. Separation of the urine will not always indicate the functional activity of the diseased kidney, so that here, again, an exploratory incision is required. However, in this case it should be made in the lumbar region, because a laparotomy does not expose the neoplasm.

Some cases of renal neoplasm will have a very rapid course, while there are others of undoubted malignancy which have remained stationary for years; these are those where metastases occur early, while others, in spite of the enormous size of the growth with numerous extensive areas of softening, have remained within the capsules, and have given rise to no systemic involvement.

Especially among the renal growths which occur in childhood there are very typical symptoms—an almost imperceptible development of the growth until an increase in the size of the abdomen calls attention to the condition. There sometimes is a regularly intermittent hæmaturia, while at others the urine constantly contains blood; emaciation is rapid, and finally severe diarrhœa occurs. In other cases quite the contrary condition arises, in which hardly any evidence of a renal enlargement can be detected, while the symptoms are those of a gastric or intestinal catarrh.

The mobility of the growth also is variable; it may be firmly adherent, while in other cases it can be grasped like an ovarian tumour, and be pushed from side to side within the abdomen.

Growths of the right kidney may be mistaken for hepatic tumours, but an enlargement of the liver can be differentiated from renal neoplasms in that the former neither extend high up into the thorax, nor do they displace the liver to any considerable degree. Generally the hand may be inserted between the costal border and the enlarged kidney—a thing which cannot be accomplished when a hepatic growth is present. In

a large majority of cases the large intestine lies in front of the enlarged kidney, which is made evident by percussion; while only in exceptional cases, either from adhesions with the gut or malformation or atrophy of the liver, no intestine will be found lying over its anterior surface. In cases of hepatic neoplasm there are usually symptoms of biliary retention, and all renal symptoms are lacking. A tumour formed by a distended gall-bladder is characterised by its pear-shaped form, and may be distinguished from an enlarged kidney by the absence of extensive dulness in the lumbar region, and by the presence of hepatic symptoms.

It is a more difficult matter to diagnose a renal growth when the latter is adherent to the liver, because the important diagnostic evidence obtained by palpation and percussion must, of necessity, give a negative result.

The absence of mobility of renal tumours during respiration is by no means a reliable sign, because very large kidney tumours have been observed to follow the respiratory movements in a most evident manner. The intestine may also be wanting in some exceptional cases over the anterior aspect of the renal growth, and, consequently, it is evident that there is not a single symptom upon which one may depend, so that in all difficult cases a careful consideration of the symptoms presented, as well as an exact history, will be of very great importance.

The left kidney, when enlarged, may be mistaken for an enlarged spleen, and here also, when there is adhesion between the two organs, the diagnostic difficulties become very great. However, a splenic tumour usually runs parallel with the costal border posteriorly, and an indentation can usually be found, while its borders are rounded. It lies flat directly under the abdominal wall, while the intestine lies behind and below it, and the history of the case and blood examination will usually settle the diagnosis. An enlarged left kidney extends downwards and anteriorly, is slightly movable, and in most cases is covered by the descending colon or the sigmoid flexure, while percussion will reveal a tympanitic zone between the spleen and the kidney.

A marked distension of portions of the colon, either on the right or the left, caused by gas or fæcal masses, may also, at first sight, resemble a renal tumour, but careful palpation will alone suffice to diagnosticate the real condition. The symptoms, likewise, point to marked disturbances of the intestinal functions, and renal symptoms are lacking.

A psoas abscess frequently occurs in the lumbar region, and from here extends into the abdomen, and may show an indistinct fluctuation or even none at all. However, a careful examination will reveal the spinal origin of the affection.

Interstitial or subperitoneal tumours of the uterus present no difficulty in diagnosis, but a very greatly enlarged kidney has been mistaken for a pregnant uterus in some few cases.

As a rare lesion I would mention malignant disease of the renal lymph-nodes and those of the pre-lumbar region, which from their rapidly increasing size may simulate malignant disease of the kidney.

When a renal tumour is found it is not always an easy matter to diagnose its true nature; thus, renal calculus, complicated by a suppurating process, causes a considerable enlargement of the kidney. A severe tuberculous pyelonephritis may suddenly develop in an infected kidney, causing very considerable enlargement of the organ, and later on cystic changes arise in the cortex of the organ.

In favour of the diagnosis of renal carcinoma we will have a relatively rapid development, which may be somewhat irregular in its progress, a firm, slightly movable tumour, which occasionally may present fluctuation in some spots, painful on pressure, with an uneven and occasionally nodular surface. Hæmaturia, as I have said, is a frequent symptom, but when this does not occur microscopic blood can usually be found in the urine. Continued pain in the renal region and a progressive decline of the general vitality, finally leading to cachexia, completes the clinical picture.

Pyelitis and pyelonephritis often cause enlargement of the kidney very similar to that found in malignant disease of the organ. The microscopic changes in the urine, without any evident hæmaturia, combined with the general symptoms of a pus-focus, are sufficient to put one on the right road for a diagnosis.

Hydronephrosis is even, rounded, and smooth in surface, usually distinctly fluctuating, giving rise to slight pain or none at all.

A paranephritis is a painful, fluctuating tumour, particularly evident in the lumbar region, while the skin covering it is generally œdematous.

Nephrolithiasis rarely gives rise to enlargement of the kidney unless complicated by a suppurative process, but microscopic blood and pus may always be found in the urine.

Simple cysts of the kidney rarely cause much enlargement, but when they do the symptoms are similar to hydronephrosis. Hydatid cysts of the kidney produce considerable enlargement; fluctuation is not often present, but the thrill peculiar to this disease will usually be found. A positive diagnosis is only possible when, from rupture of one of the sacs, hydatids may be found in the urine.

Hæmorrhage into the renal parenchyma, calices, and renal pelvis may cause considerable enlargement. In these cases the history will often explain the cause of the renal enlargement, but the possible causal connection between such hæmorrhages and the development of a malignant growth must always be recalled.

September 19th, 1910.

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CONTENTS.

PAGE

*Clinical Lecture on Acute Bulbar Paralysis (Polio-encephalitis Inferior) in a Girl aged thirteen years. By LEONARD GUTHRIE, M.D., F.R.C.P. 385

*Innocent Tumours of the Rectum. By C. GORDON WATSON, F.R.C.S. ... 389

*Demonstration of Cases at the Medical Society of London 394

Severest Anæmias 400

**Specially reported for the Clinical Journal. Revised by the Author.*

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CLINICAL LECTURE

ON

ACUTE BULBAR PARALYSIS (POLIO-ENCEPHALITIS INFERIOR) IN A GIRL AGED THIRTEEN YEARS.*

By LEONARD G. GUTHRIE, M.D., F.R.C.P.

SOME of you will remember that I showed this patient at a demonstration held at the Post-Graduates' College on March 1st of the present year.

The case is important and instructive in many ways, and you will probably be glad to see the patient again and to observe the progress which has been made since.

The girl is now nearly fourteen. She is small and undeveloped for her age. She has always been delicate, and at the age of seven she had otorrhœa, which subsided in three weeks and has not returned.

The history of her present illness is as follows: On January 13th, 1910, whilst getting up she complained of frontal headache and vomited. Four hours later she became suddenly dizzy and fell down as if faint. Her eyes wandered and stared, the jaws were rigidly closed, she foamed at the mouth, and was unconscious for a few minutes. She then recovered consciousness, but was speechless, unable to swallow, and completely paralysed from head to foot.

On admission to hospital on January 17th she was a thin, ill-nourished girl. The temperature was 100·2° F., the pulse small, regular, 120; respirations 42; heart-sounds normal.

She was quite conscious, judging from the expression and movements of her eyes, but could not speak, swallow, or move head, trunk, or limbs. The pupils were equal, reactions normal. There was no paralysis of the ocular muscles, but very slight ptosis of the left eyelid.

* Delivered before the Post-Graduates' College and Polyclinic.

The lower half of the left side of the face was distinctly weaker than the right. The orbicularis oris was flabby and motionless, the lips were everted, and saliva constantly dribbled from the mouth. She could not purse her lips or blow. When crying the levator anguli oris and zygomatici only were in action. There was slight trismus, so that the jaws could not be separated more than an inch apart. She could not protrude or move her tongue; the soft palate was motionless on stimulation.

When milk was placed in her mouth no movements of swallowing occurred, but the fluid caused immediate choking, and loose, weak, ineffectual cough suggesting imperfect apposition of the vocal cords.

There was complete motor aphasia. She moaned and cried, but could produce no articulate sound. The neck muscles were powerless; she could not raise her head from the pillow or move it from side to side. She could not sit up unsupported or turn over.

The intercostal muscles and diaphragm were slightly over-active; the abdominal muscles acted normally. On auscultation loud, coarse râles, due to accumulation of mucus in the larger bronchi, were heard all over the lungs.

All four limbs were in a condition of complete flaccid paralysis. When lifted out of bed and suspended by her armpits her head fell forwards, and she hung as a dead weight, with feet trailing on the floor.

The abdominal reflexes were present, the knee-jerks absent. Plantar responses were negative, but on tickling the soles of the feet the legs were drawn up in involuntary spasm. Sensation seemed normal everywhere. She cried when pricked or pinched in any part. There was complete incontinence of urine, and the bowels were confined.

Her condition was obviously one of extreme gravity. She had taken no food for four days, and being quite unable to swallow was fed at once by nasal tube. Strychnine and atropine were injected subcutaneously every four hours.

The temperature ranged from 100·2° to 102·2° F. for four days, and then fell to normal.

In two days after admission there were signs of general improvement. She gradually regained power first in the left leg, secondly in the right leg, and thirdly in the left arm, but the right arm was still flaccid and completely paralysed.

The trismus and ptosis noticed on admission soon passed off, and the paresis of the left side of the face was less marked than it had been. By January 27th she could move her head freely and raise it from the pillow. She was still speechless; the orbicularis was paralysed. She could not suck, or purse her lips, or swallow, and feeding was entirely by nasal tube. The tongue was wrinkled and beginning to waste, but she could protrude it just beyond her teeth. The soft palate was still motionless. The cough was much more forcible; the râles in the lungs had almost disappeared. She moved both legs and also the left arm freely, but the right was still flaccid and paralysed. The triceps and wrist-jerks were exaggerated, the knee-jerks absent. Incontinence of urine persisted, and the bowels only acted after enemata.

By February 15th there was general improvement in muscular power. She could sit up without support, and could move the right shoulder and elbow, but the wrist and hand remained paralysed. The orbicularis oris acted fairly well, and she could protrude the tongue well beyond the teeth.

On this day she was able to swallow milk for the first time for nearly six weeks, and nasal feeding was discontinued.

The incontinence of urine ceased on February 17th, and on the 24th she was able to get out of bed and swallow solid food.

On March 1st, when you last saw her, she was, as might be expected, considerably emaciated, but there was no local wasting of any muscle groups. She could stand and walk a few steps if supported on either side. The right hand and wrist were still useless, and the tendon-jerks exaggerated, but there was no rigidity about the limb.

Although the movements of tongue and lips seemed practically normal, she was quite dumb, and only able to vocalise vowel sounds—"*ah*" and "*oh*." The soft palate was immobile, but at no time had there been regurgitation of fluid through the nose on deglutition.

Mentally she was quite intelligent, though emotional, crying or laughing on slight provocation. Even at the height of her illness she always contrived to make her wants known by signs.

The constant dribbling of saliva from the mouth created a false impression of mental weakness.

Present condition.—She has gained many pounds in weight and is plump and well nourished though

she is small, and her development is that of a much younger child. Her disposition is cheerful and obliging, and she makes herself generally useful to the nurses and patients in the ward. Muscular power is fully restored in the lower limbs, but in walking the movements of the right leg are slightly spastic. The plantar response in the right side is extensor; in the left it is negative or flexor. The knee-jerks are still absent even on reinforcement.

The left upper extremity is normal. Movements of the right shoulder, elbow, and wrist are free, but those of the right hand are still weak. She can, however, write fairly well with the right hand, and also knit.

The wrist and elbow-jerks are exaggerated on the right side. Rigidity, ataxy, and tremors are absent.

The upper halves of the face are normal, but there is still slight weakness of the lower half of the face on the left side—noticeable when she shows her teeth or tries to blow, but not when smiling.

The mouth is not now flabby and half open, and the constant dribbling of saliva from it has ceased.

The tongue can be protruded to its full extent and is not wrinkled or wasted.

The soft palate is, however, motionless on pulsation. On laryngoscopical examination the vocal cords are seen to act normally.

Electrical examination.—Dr. Wilfrid Harris, my friend and colleague, has kindly tested the reactions of the tongue, palate, and orbicularis oris. All respond normally to faradism and galvanism. The pharyngeal reflex is readily obtained.

Speech.—During the past two months power of speech has been gradually restored, but there is still well-marked motor dysphasia or dysarthria of the bulbar type.

The voice is low-pitched, weak, and nasal.

She speaks in expiratory gasps, air escaping from the nostrils with each utterance of words owing to inaction of the soft palate.

The labials, linguo-dentals, anterior and posterior linguo-palatals are all indistinct or unpronounced.

The dysphasia is, however, motor in the strictest sense. The difficulty is purely in articulation. Speech is fluent though hard to understand. In motor aphasia of cerebral origin, the defect is very rarely in articulation alone.

Articulation may be faulty in such cases, and many cannot utter a word at will. Yet in most, the reiteration of some word or short sentence when any attempt at speech is made shows that the motor mechanism of articulation is unaffected. For instance, a patient at present in hospital says only "Then, besides," and another says, "Praise the Lord!" whenever she tries to speak. Obviously aphasia in such cases is not due to inability to utter words but to initiate them. Motor aphasia of cerebral origin is usually complicated by verbal amnesia and by word-deafness or word- and letter-blindness (sensory aphasia) and agraphia.

Our patient has no form of sensory aphasia, but simply a defect in articulation. She names objects readily, understands spoken and written words, and can write spontaneously or at dictation and copy correctly.

We have now to localise a lesion which produces, first, slight crossed hemiplegia, namely weakness of the lower half of the left face and of the right arm and leg, with exaggerated wrist- and elbow-jerks and extensor plantar response in the affected limbs, and second, dysarthria or defective articulation.

In the first place, the crossed hemiplegia is of cerebral type, as shown by the exaggerated reflexes and absence of local muscular wasting in the right arm, and by the fact that the weakness is of the lower half and not of the whole of the left side of the face.

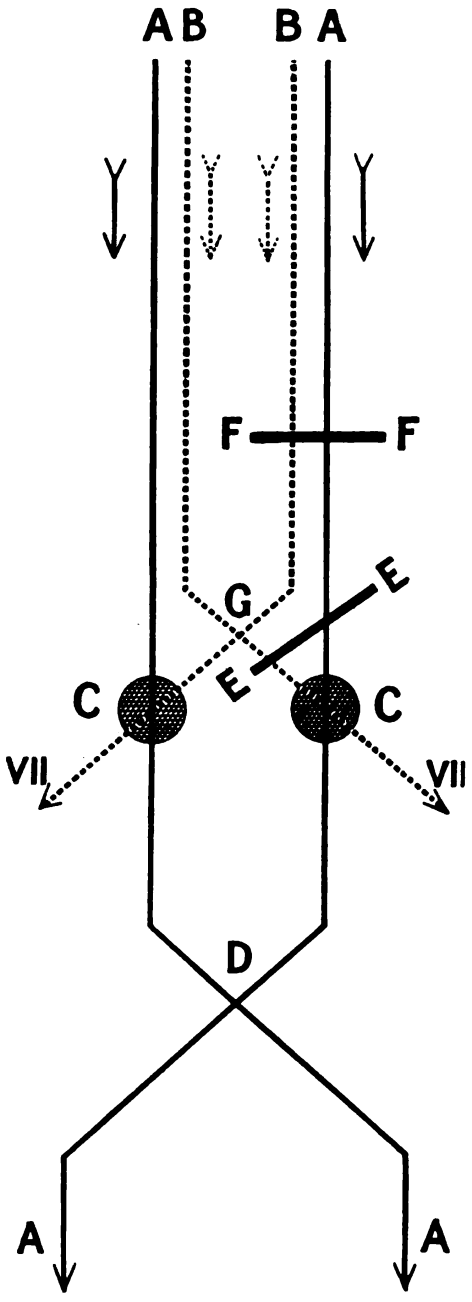
I may remind you that crossed hemiplegia in which the face is completely paralysed on one side and the limbs on the other is due to a pontine lesion, which involves the pyramidal fibres before their decussation in the medulla, and either the nucleus of the seventh nerve or the fibres of the seventh nerve itself on the side of the lesion.

But in such cases the facial paralysis is of peripheral type, involving the whole of the facial muscles. Moreover, when nucleus or nerve is destroyed the electrical reactions suffer too, whereas in the present case they are normal.

Hence we must assume that the lesion must be above the nucleus of the seventh nerve, yet in such a position as to damage the motor tract to the right side of the body and the nerve-fibres leading to the seventh nucleus on the left side.

A lesion in the upper part of the pons just above the facial nucleus on the left side would probably cause facial paralysis of the supra-nuclear type on

the left side and hemiplegia on the opposite side, as indicated in the diagram (*cf.* diagram).



Drawing to show the mechanism of crossed paralysis and probable site of lesion in the present case. A A. Motor tracts. D. Decussation of motor tract in medulla. B B. Path of facial fibres from hemispheres. G. Decussation of facial fibres in pons. C. Nuclei of seventh nerve. VII. Seventh nerve after leaving nuclei. A lesion at F causes ordinary hemiplegia of the opposite side. A lesion at C paralyses the whole of the facial nerve on the one side and the fibres for the arm and leg (A) on the other. A lesion at E just above the nucleus will cause paralysis of the lower half of face (supra-nuclear type) on one side and of the arm and leg on the other.

A lesion in this position would again account for the dysarthria, as it would interrupt not only the motor path to the limbs on the opposite side, but also the motor fibres leading to the nuclei in the medulla, which govern the muscles of articulation and the structures innervated from the bulbar centres.

Needless to say, I am only attempting to localise a lesion which explains the symptoms which exist at the present time. The history of the illness shows that originally the lesion must have been far more extensive than it is now.

The paralysis at the onset involved the neck, trunk, and all four extremities, besides the muscles concerned in speech and deglutition. Irritation of the nucleus of the motor branch of the fifth nerve was indicated by the presence of trismus during the first few days, and the ptosis of the left eyelid noticed at the same time shows that the ocular nucleus in the mid-brain must have been slightly affected. Had the vital centres governing heart, circulation, and respiration been seriously damaged, the result, no doubt, would have been fatal.

The absence of knee-jerks and incontinence of urine are worthy of comment, though difficult to explain. One can only assume that the incontinence was caused by disturbance of the lumbar centres. The persistent loss of knee-jerks cannot be attributed to affection of the anterior cornual cells or the motor nerves which proceed from them, as there is no local wasting nor weakness of the quadriceps muscles on either side.

The lesion must therefore be either on the sensory side of the reflex arc which is concerned in the production of the knee-jerk, or affect the lumbar nuclei themselves. It is true that signs of involvement of the posterior columns such as loss or alteration of sensation, ataxy, and hypotonia are absent, but loss of knee-jerk may be the solitary symptom of disease in ascending tracts of the spinal cord.

Nature of the lesion.—The sudden onset of the illness points to an acute bulbar lesion, which at first produced widespread paralysis of the trunk and limbs and of the parts innervated by the lower part of the bulb. Such a lesion might be vascular or inflammatory.

Hæmorrhage may be excluded and also vascular occlusion, in the absence of any disease such as syphilis, nephritis, or morbus cordis.

The possibility of diphtheritic paralysis was entertained and rejected in the absence of any history of sore throat. It is true that sudden and sometimes fatal "bulbar crises" may occur in diphtheritic paralysis, but they are always preceded by minor signs of that affection such as squint, nasal voice, regurgitation of fluids, weakness of the limbs and loss of knee-jerks.

A diffuse polyneuritis due to alcohol, lead, arsenic or any form of toxæmia is negatived both because none of the usual causes was present and also because paralysis due to polyneuritis is always gradual in onset—never sudden.

For a similar reason the rare form of Landry's paralysis, in which the upper parts of the body are affected by motor paralysis without loss of sensation, may be excluded. Moreover, the affection of speech and partial hemiplegia of upper neuronic type are against the diagnosis. Landry's paralysis is never quite sudden in onset.

The history and course of the illness are quite unlike that of paralysis due to a bulbar new-growth.

The pyrexia—sudden onset and diffused symptoms—are in favour of an inflammatory lesion, and polio-encephalitis inferior is the most probable diagnosis.

As I have already mentioned, the normal electrical reactions show that the bulbar nuclei have not been damaged. But polio-encephalitis inferior does not necessarily attack the nuclei alone any more than polio-myelitis affects solely the anterior cornual cells. As Pfaundler and Schlossman ('Diseases of Children,' vol. iv) observe: polio-encephalitis inferior may involve the pyramidal tract, causing hemiplegia, monoplegia, besides producing "disturbance of speech, dysphagia, interference with movements of mouth and tongue, aphasia, disturbance of pulse and respiration, dribbling of saliva, hysterical laughing and crying," and this description applies fairly well to the case now under consideration.

The prognosis in our patient's case is eminently favourable. Steady progress is being made, and there can be little doubt that in time recovery will be complete.

With regard to treatment, I need only say that nasal feeding and careful nursing saved the patient's life. During the first few days following admission to hospital, the accumulation in the air-passages of mucus which the child was unable to cough up or expectorate threatened suffocation, but the symptoms soon subsided under the use of injections of strychnine and atropine, a mode of treatment which has sometimes been successful in the similar condition present in severe cases of diphtheritic paralysis.

September 26th, 1910.

INNOCENT TUMOURS OF THE RECTUM.

A Post-Graduate Lecture delivered at St. Mark's Hospital.

By C. GORDON WATSON, F.R.C.S.,
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In the surgery of the rectum and anus, innocent tumours, outnumbered and overshadowed by their malignant brethren, play but a minor part. It follows, therefore, that the task which devolves on me to-day must of necessity present difficulties, which are inevitable to one of limited experience. I hold that lectures are only of value in so far as they deal with personal clinical experience. It would ill become me, and would be no compliment to you, to deliver a text-book lecture on the varieties, symptoms, diagnosis, and treatment of benign tumours of the rectum. In far less time, and with far more profit, you could acquire this information for yourselves. I shall therefore content myself with the relation of a few cases that have come under my personal observation, and endeavour to extract some points of interest from them.

The interest which attaches to the simple tumour of the rectum would be small were it not that, as in other parts of the body, the dividing line between innocency and malignancy presents difficulties which must often arise both in diagnosis and treatment.

During the past two years more than one case has come under my notice of a benign tumour overstepping the bounds of innocency and terminating in malignancy.

A man, æt. 65 years, came to the out-patient room in the summer of 1908 with a simple polypoid adenoma of the rectum. This I removed by transfixion after dilating the sphincters, and the patient returned to the country. A year later he returned to the hospital complaining of pain, the passage of blood in his stools, and of having lost flesh rapidly. On examination I found just within reach of the finger at the site of the original polypus an ulcerating growth of the rectum, which was fixed beyond hope of removal. I performed an inguinal colotomy. Subsequently he developed symptoms of obstruction, and on digital examina-

tion by the colotomy wound a second growth could be felt in the descending colon above the colotomy opening. I again opened the abdomen and performed a transverse colotomy, verifying the existence of the second growth in the descending colon. The man eventually died at home, so that we had no opportunity of a post-mortem examination. But I think there is little doubt that this was a case of multiple adenomata of the large bowel, developing into carcinomata. I have met with a somewhat similar case in the post-mortem room at St. Bartholomew's.

The specimen which I now show you is another example of the combination of innocency and malignancy, the latter condition undoubtedly a development of the former. The specimen is a portion of the pelvic colon, which I resected more than three years ago at the Metropolitan Hospital. The patient was æt. about 40 years, and a sergeant of police. He suffered from symptoms of colitis, frequent stools, often blood-stained, wasting, and general malaise. On examination under an anæsthetic I detected a small lump in the left iliac fossa. With the sigmoidoscope I ascertained that the lower ten inches of the bowel were normal. I explored the abdomen and found a movable tumour involving the pelvic colon. This I resected with some ten inches of adjoining bowel, and performed an end-to-end anastomosis. On opening the resected gut I found that the surface of the bowel was studded with multiple polypoid adenomata. In the midst of these innocent tumours lay the offending growth, an adeno-carcinoma, which was rapidly constricting the gut, and which would have caused acute obstruction in the near future. The man made an excellent recovery, returned to the police force, and still continues well.

This case, I think, explains the first case which I related, and demonstrates the development of a carcinoma from a simple adenoma, and the specimen is practically identical with another specimen in the museum at St. Bartholomew's. I have related these cases to show that as in other parts of the body, a tumour of the rectum, which begins life in all innocence, may end in malignancy. This is important to remember from the point of view of prognosis, and it is important to remember in considering treatment. The specimen I now show is an innocent papilloma of the rectum, which I removed last year. You will see that it is a pedunculated tumour, and that I have removed it fairly freely as regards the mucous membrane at the neck. This I did by transfixion and ligature. I would like to emphasise the importance of transfixion in removing pedunculated tumours, because unless this is done considerable risk is run of severe hæmorrhage should the ligature slip. Quite large vessels may be seen supplying these tumours. Unless transfixion is employed it is not possible to remove a free margin of mucous membrane without incurring considerable risk of the ligature slipping off.

Another point I would like to emphasise is the importance of a microscopic investigation of all tumours, however innocent they may appear. A year or so ago a medical man was involved in legal proceedings of a very serious nature, because it was alleged that he had performed an operation of unnecessary magnitude on a tumour of the rectum, under the assumption that the tumour was malignant, when in reality it was innocent. Fortunately for this medical man the specimen had been preserved, and could be submitted to experts who confirmed the malignancy.

Treatment is necessarily largely influenced by the microscopic evidence, which may be quite at variance with the naked-eye evidence. Recently, at St. Bartholomew's, I saw a section of a small inflammatory tumour, which had been removed from the rectum. The history of the case was as follows: An ordinary rectal polypus had been ligatured and removed; at the site of ligature a fibrous-looking tumour had subsequently appeared, which was regarded as a recurrence. This was removed locally, and showed only inflammatory tissue under the microscope. Removal was again followed by recurrence, and a serious doubt arose as to whether a mistake had not occurred in the original diagnosis. However, the final section explained the case. The tissue removed was undoubtedly chronic inflammatory tissue, and giant-cells were present, which were suggestive of tubercle. A careful search, however, under the microscope revealed a small piece of silk ligature in the section, and around it several giant-cells. The silk ligature, having started a chronic inflammation, had become embedded, and the cause of irritation remaining, a fibrous tumour had resulted. If silk is used for ligatures in the anal canal, then it is advisable to leave the ends long as is customary in

the ligation of piles. It is then possible to be sure, when the ligatures have separated, that no silk remains behind to give rise to irritation. Fine silk may be used for the ligature of small vessels without any fear of its giving rise to trouble, but thick silk, if used in the anal canal, should always be left long.

Except in ligaturing hæmorrhoids, where strong silk is essential if the vessels are to be securely tied, I prefer to use catgut in this region. I always use catgut for Whitehead's operation, though sometimes with the addition of a few supporting silk sutures to prevent separation at the muco-cutaneous junction. I might perhaps mention here that some separation at the line of junction in this operation always occurs during healing, but it is only wide and complete separation that is harmful.

I have been led to a digression on ligatures in considering the treatment of a simple innocent tumour in the rectum. The specimen which I have just shown you was about three inches from the anus. It was necessary to thoroughly dilate the anus before attempting its removal. Some care was then required to get at the tumour for the purpose of transfixion. I mention this because if the tumour is seized with volsellum forceps and then pulled down some tearing of the mucous membrane may occur, giving rise to hæmorrhage, which is very difficult to deal with. Considerable care is also required in dilating the anus. If any force is used, except gradual force, or if the patient is not fully under the anæsthetic, then it is easy to tear some of the sphincter fibres, with the possible result of loss of sphincteric control for several weeks. The sphincters appear to be more intolerant of rupture than of division with the knife without laceration. It is commonly supposed that if the external sphincter is divided in more than one place that incontinence follows. This need not be so. Recently in a very extensive and complicated case of fistula I found it necessary to divide the sphincter in three places in order to freely open all the tracks. No harm resulted and perfect control was speedily restored with the healing of the tracks.

In dealing with innocent tumours at the anus and in the anal canal, such as anal papillomata and villous tumours, great care is required not to damage the sphincters and to avoid subsequent stricture or ulceration through leaving a raw granulating surface. Consequently it is advisable to proceed in such cases on the same lines as in Whitehead's operation and not to attempt removal of the tumour only, but rather to remove a complete circle of mucous membrane by dissecting up the membranes of the anal canal freely, and so freely that the mucous membrane of the rectum above can be brought down to the anus and held there by stitches *without tension*. Any tension is fatal to sound healing without cicatrisation, as it is in Whitehead's operation for hæmorrhoids.

In the diagnosis of tumours of the anus and the rectum, as I have already stated, we naturally rely on the microscope to differentiate the innocent and malignant, but it is well to remember that the microscope may fail to show a malignancy which really exists owing to the fact that the portion of the tumour which is cut does not include the base. This is especially true with regard to villous tumours of the rectum. Little difference exists if only the surface villi are seen, and it is only when the basement membrane of the mucous surface is seen to be invaded and pierced by columns of cells that the malignancy is apparent.

The specimen I now show illustrates a papilloma becoming malignant and associated with a fistula. The papilloma is of unusual size and was situated just within the anus. The greater portion of the tumour is innocent, but at one part of its base it is becoming malignant and undergoing colloid degeneration. This portion is at the neck and close to the skin of the anus. A small fistula connects the cancerous portion with the exterior and is indicated by a glass rod.

Recently we had a case in the hospital here of a large growth of the rectum necessitating colotomy. The case presented some unusual features, especially as regards the length of history. On more than one occasion a portion of the growth was removed for section and showed no evidence of malignancy. Ultimately, and this I believe to be a rare event in malignant disease of the rectum, several nodules appeared beneath the skin of the abdomen. They appeared one at a time, starting near the umbilicus, as small, pin-head nodules and spreading centrifugally. At intervals of a few days further nodules appeared, more remote from the umbilicus and nearer the costal arch. One of these was cut and found to be a round-celled sarcoma. The metastasis had apparently spread

along the lymphatics of the umbilical cord to the skin of the abdomen, and so onwards in the fascial planes beneath the skin in the same way that carcinoma of the breast sometimes extends. But for the presence of these secondary nodules a definite diagnosis would not have been made. I mention the case, because in the opinion of some who saw the patient the tumour was considered to be a gumma.

Another point which I should like to call your attention to is the association of inflammatory mischief with new growth in the rectum. A few months ago a man came to the out-patients' with a large ischio-rectal abscess, suffering great pain and feeling very ill. He was at once admitted, and it was then found that he had an inoperable carcinoma of the rectum. Invasion of the growth through the rectal wall and a secondary infection had led to an abscess which might easily have allowed the growth to escape attention. Mistakes are so easy to make and so easy to criticise. I can imagine this patient having come to my out-patient department when there were no empty beds. I might have incised the abscess as a temporary measure, and told the patient to return for daily dressing until such time as there was a vacant bed, without having diagnosed the gravity of the case. Then I can imagine that, tired of waiting, he might seek admission elsewhere and bring down on my head some sharp criticism when the malignant disease was discovered. I think it is a good rule never to omit the rectal examination even in the most obvious cases of external complaints, and especially with patients who complain of bleeding and are past middle life. I remember once seeing a post-mortem of a man who died from general peritonitis following on perforation of the rectum due to carcinoma just beyond reach of the finger. The patient had complained of rectal bleeding and suffered from hæmorrhoids, for which he was duly treated by ligature. About a week or so later, when recovering from the operation, sudden severe abdominal symptoms occurred, which were very puzzling as a sequel to the ligature of piles. It was thought that perhaps there had been local sepsis followed by a septic thrombosis of a large vein within the abdomen, and many other things were thought of except the actual state of affairs. In passing I would remind you that a septic thrombosis of an iliac vein gives rise to symptoms which closely simulate peritonitis, but with this difference, that rigidity of the abdominal muscles is not very likely to be present in thrombosis and not very likely to be absent in general peritonitis. Again, in passing, may I say, with regard to ligature of hæmorrhoids, more especially as there has been some correspondence in one of the journals lately on the subject, that the operation of ligature is *not* always free from complications. The most serious result which I have seen occurred in South Africa during the war. A yeoman, after operation for piles, developed a septic pylephlebitis, followed by an abscess of the liver, from which he eventually recovered after a very prolonged convalescence.

The specimen, which I now show you, of the lower six or seven inches of the rectum, which I excised about two months ago, illustrates the following important dictum, that innocent tumours of the rectum of a certain size in a certain situation require to be treated as if they were malignant. The tumour is a villous papilloma involving two thirds of the circumference of the rectum and involving about three inches of its long axis. It is situated in that part of the rectum which is in direct relationship with the prostate and base of the bladder. There is no ulceration of the surface, and no constriction of the lumen of the bowel, nor has the growth extended through the muscle wall. I excised the lower six or seven inches of the rectum by removing the coccyx and exposing the bowel from behind by an incision extending from just behind the anus to the sacro-coccygeal junction. The peritoneum was opened on either side of the bowel, and the rectum freed all round. The patient was then placed in the lithotomy position, and an operation commenced corresponding to the first stage of a "Whitehead," the lining membrane of the anal canal being dissected free of the sphincters. The muscular wall of the rectum was then cut through above the level of the sphincters, and the bowel with the growth drawn through the anus and cut through well above the growth. The proximal cut edge was then sutured to the skin of the anus, and a drainage-tube with gauze packing passed into the sacral incision.

This may seem somewhat of a severe operation for an innocent tumour, but experience shows that these tumours frequently become malignant, and commonly recur if not freely removed. No operation short of an excision could secure removal

of this growth and leave the lumen of the bowel intact. The growth was situated too low down to allow of removal and an end-to-end anæstomosis, and experience shows than an anastomosis low down practically always gives way posteriorly, and results in a fæcal fistula.

The operation which I have described is well suited to cases of this kind, and should, in my opinion, be reserved for this class of case, and not used in preference to the combined abdomino-perinæal route for malignant disease. In all cases of malignant disease involving the anal canal, rectum, or descending loop of the pelvic colon, which are not definitely "inoperable," the abdomen should be opened. A complete investigation can then be made of the extent of the growth and its relation to the bladder, etc., of the condition of the lymphatic glands and the liver. A decision can then be arrived at as to the possibility of operating, with a view to a cure of the disease, or as to the necessity of abandoning any attempt at removal, and being content with colostomy. In this way attempts to perform radical operations by the sacral or perinæal route are prevented in many cases, in which the full extent of the disease cannot be ascertained until the abdomen is explored. If a radical operation for malignant disease is feasible and desirable, it can only be performed by a combined abdomino-perinæal method, because only in this way can the upward spread of the disease be prevented (if preventible) by removal of the lymphatic vessels and glands in the meso-sigmoid and along the inferior mesenteric vein, together with many inches of the bowel above the growth.

In the case of innocent tumours, such as the present one, which is on the border-line of malignancy, it is probably unnecessary to remove the lymphatic glands, and therefore an operation from the perinæum is both suitable and desirable. For the simple polypoid papilloma, local removal; for the more advanced sessile villous papilloma, sacro-anal or perinæal excision; for the malignant tumour, abdomino-perinæal excision should, I think, be the rule.

The term "polypus" is used to denote any pedunculated tumour, and covers the adenomatous polypus so common in children, as well as the villous tumour and the fibrous polypus. The adenomatous polypus arises in hypertrophy of the glandular tissue of the crypts of Lieberkuhn, and is usually met with in the lower two inches of the rectum above the anal canal, and occasionally higher up, as in the specimen I now show. At first sessile and small, it gradually enlarges and becomes pedunculated when dragged on by the passage of fæces. Eventually the stalk becomes sufficiently elongated to allow protrusion of the polypus through the anus. Until this happens it may give rise to no symptoms except to an occasional blood-stain in the motions. When the polypus comes within grasp of the sphincters, then attacks of straining and a blood-stained discharge occur. They are rarely met with in adults. It seems probable that, considering how frequently they are met with in children and how seldom in adults, that they sometimes get torn off during defæcation and passed unknown, so that a spontaneous cure results.

Mention must be made of the hypertrophied papillæ, so often met with round the anus, especially in the presence of a rectal discharge which keeps the skin sodden and septic and gives rise to chronic inflammation. These hypertrophied papillæ or anal papillomata are often met with in clusters, and are identical with those met with round the vulva and on the penis in cases of gonorrhœa (gonorrhœal warts). Gonorrhœa is not necessarily the cause. They are easily removed by snipping through each papilla at the base, checking hæmorrhage with a little adrenalin or the electric cautery, and dusting on iodoform, but they will recur unless the rectal discharge is cured. A diagnosis will sometimes have to be made between papillomata and anal epithelioma. In papillomata each papilla is distinct and separate and the skin in between the papillæ is normal. In epithelioma this is not so: the growth is continuous and sessile.

Fibromata and lipomata are also met with round the anus. Some years ago I removed quite a large fibro-lipoma from the ischio-rectal fossa under a local anæsthetic, but this is the only case of the kind I have met with.

Of rare conditions I may just mention post-anal and rectal dermoids. A few months ago I met with a case of a post-anal dermoid. The patient stated that a small abscess had been opened behind the anus and would not heal until quite recently, when the sinus had closed and the abscess had re-formed. I found that there was a post-anal dimple or sinus along which a probe passed for about half an inch towards the coccyx, and then

stopped. This sinus was skin-lined, and immediately behind this, lying over the tip of the coccyx, there was a small subcutaneous swelling the size of a hazel-nut, and over it the scar of the old incision and sinus. This I cut into, and to my surprise a curl of dark hair and some cheese-like material came out. Clearly this dermoid cyst was at one time the termination of the blind post-anal dimple that I have described.

The specimen I now show is a large dermoid cyst removed from beneath the mucous membrane of the rectum of a woman, æt. 39 years. During a confinement the tumour was protruded through the anus by the descent of the head. The pressure of the head caused rupture of the mucous membrane and the cyst was then easily enucleated. The cyst contains two hair-balls, and at the time of removal contained much sebaceous material.

I have referred to inflammatory tumours in the rectum resembling growths, and I should like to mention a case which was a little puzzling at first. A man came complaining of pain on defæcation and a small lump in the anus. I found that he had at the anal margin a small, hard, somewhat irregular nodule about the size of a marble, which did not appear to be inflammatory, but rather suggested a fibroma or fibro-sarcoma, so I removed a small portion for microscopic examination, which was, as it happened, lost. The patient appeared again the following week, and then we found that this lump was concealing the entrance to a blind internal fistula, and was merely a mass of inflammatory tissue which possibly had formed around some foreign body which had started the fistula. I have on several occasions been able to trace the internal opening of a fistula by feeling beneath the mucous membrane an inflammatory nodule similar to the one described.

The specimen now shown is a papilloma of the rectum produced by *Bilharzia hæmatobia*. The polyp consists of numerous dense papillæ, and its substance is filled with the ova of the parasite. There were several other smaller polypi in the rectum. The specimen shown prolapsed during defæcation and was ligatured and removed from a boy, æt. 14 years, who had suffered from diarrhœa and rectal hæmorrhage for some time (in Alexandria).

May I conclude this lecture by relating to you a case of rare "tumour in the rectum," a "tumour" innocent enough in itself, yet fatal to the patient, which occurred at St. Bartholomew's when I was a student. A patient came complaining of a lump in the rectum. The house-surgeon made the diagnosis of a "bottle in the rectum"; for an hour or so he did his best to remove it, and eventually sent for his senior, Mr. Alfred Willett. Under an anæsthetic Mr. Willett set to work with craniotomy forceps, and although he several times succeeded in gripping the bottle, at the critical moment of extraction the bottle slipped. The assistant surgeon, Mr. Harrison Cripps, then tried his hand and failed. Finally Mr. Willett took post at the anus and Mr. Cripps opened the abdomen, and then by the combined method the bottle was delivered. Unfortunately the rectum sloughed and the patient died of peritonitis. The history of the case was interesting. The patient suffered from pruritus, and as Mr. Cripps relates the story, he shrewdly suspected worms to be the cause. Not being a fisherman, and yet familiar with the pots of worms which his friends took with them when they went fishing, he conceived the idea of catching his worms by introducing up the rectum a stone ginger-beer bottle lined with moss to attract them. As Mr. Cripps relates, this ignorance of fishing cost him his life.

September 26th, 1910.

DEMONSTRATION OF CASES AT THE MEDICAL SOCIETY OF LONDON.

Cases of Congenital Dislocation of Hip treated by a Modification of Lorenz's Operation.

Mr. H. Stansfield Collier presented eight cases of congenital dislocation of the hip on which he had operated by the method of Lorenz, preceded in each instance by the pinching of the obturator nerve in the pelvis. That was done a fortnight before replacement was applied, and a weight extension of some four pounds was applied, so as to stretch the paralysed adductor. Mr. Collier had employed the method fourteen or fifteen times since October, 1909. He would confine himself to the expression of a belief that the results were better than he had experienced previously, though he had to regret one case in which he failed to get reduction, and a second

case in which the dislocation relapsed, to be successful, however, on the second occasion. He had found that the amount of violence necessary for reduction had been much less, that shock and pain had been distinctly less, and the amount of hæmorrhage, as a rule, inconsiderable. He said he could not speak of the ultimate result, as he had only been doing the operation two years, but by contrasting the cases with those he had treated in former times the prospects were brighter. He would not say why he preferred to pinch the nerve, but would reserve that until his knowledge was more complete.

Cases of Congenital Dislocation of the Hip-joint after Lorenz's Operation.

Mr. H. A. T. Fairbank described three cases of congenital dislocation of the hip-joint after Lorenz's operation. In demonstrating one of the cases he said that the great point was to get the child young enough. If the case was obtained young enough, then in three out of every four cases the result should be as good as he showed. If the age was unsuitable, the result had nothing to do with the operation as such. It was simply that the leg had not grown. But even then the patients walked well after the operation. The case he demostrated was æt. 3 years when operated upon, and it was done just over two years ago. Both hips were out when the operation was done, but now they were both in. When both hips were out the result was not nearly as good as it was when only one was affected.

Mr. A. H. Tubby congratulated Mr. Collier and Mr. Fairbank on the excellent results which they had achieved. He had himself never seen any better results in the various cases he had come across. Mr. Fairbank had dwelt on one very important point, namely the age at which the patients came before one. And that seemed to be the crucial point of the treatment, that after the age of four years in bilateral cases, and after seven years of age in unilateral cases, the difficulties of satisfactory reduction by the Lorenz method or by any modification of it were enormously increased. It was about eight years ago that Lorenz published his second book on what he called bloodless reposition of congenital dislocation of the hip, and shortly afterwards he (Mr. Tubby) began the treatment of the cases, which were not as well understood then as they were now. The result was that he had a number of cases which were somewhat advanced in age to treat, and he formed a rather adverse opinion of Lorenz's work compared with the results which Lorenz himself obtained. But those cases were now fully recognised, the importance was grasped of seeing them early, and there had been the introduction of skiagraphy. The net result was that those cases were now brought to hospital at eighteen months and two years of age, and they were operated upon at once. Consequently the successes from the operative treatment of the cases had been enormously increased. The most recent article on the subject, giving the results over many years, was one by Dr. Bradford, in the American Orthopædic Society's 'Transactions,' and he considered that of the unilateral cases under the age of four, 90 per cent. ought to be cured, so that the important fact altogether was that of age. In looking at Mr. Collier's cases there was one point which struck him very much, and he had often noticed it in his own cases, namely that even when the skiagram showed the hip to be in perfect position, and the walk was as good as it could be, when one felt the head of the femur it seemed to be higher than it should be and more advanced into Scarpa's triangle, and he had often noticed that that condition was associated with another condition, namely that the outer surface of the trochanter was placed more backward than it should be. He took it that the meaning of that was that in those cases there was associated with the congenital dislocation another condition which was often in later life very inimical, namely incurvation forwards of the neck of the femur, such incurvation as one sometimes saw complicating coxa vara. The neck of the femur was convex forward, and therefore the head had not its proper relation to the shaft. In those cases he had often been considerably misled. He used to think that the head was not in, but was somewhat displaced, and he thought the head was not in because the outer surface of the trochanter had not its position with regard to the other parts of the bone. But he thought the position was due to the fact that the neck of the femur was convex forward, and in later cases that convexity forwards increased enormously the prospect of permanent reduction, because it had been shown that cases over six or seven years of age with that convexity forwards were extremely

liable to relapse unless one took great care to keep the leg fully inverted. He congratulated Mr. Collier on his idea of pinching the obturator nerve; it seemed a very ingenious and satisfactory idea, and he (Mr. Tubby) would like to hear more of it. His own practice in those cases, and he had operated upon them for seven or eight years, had been to excise a very considerable portion of the adductor muscles, sometimes as much as half an inch, but in attacking the adductor magnus, and in those cases where the adductor spasm had been very severe, he had divided the adductor magnus above its insertion into the lower part of the femur, and having paralysed the adductors he put on weight extension of 4, 5, or 6 lb., and proceeded to reduce them mainly by traction and adduction and by manœuvres advocated by Lorenz. But when cases were over eight years of age, or when the flexion had exceeded 1½ in. and the part was very resistant, he had resorted to pulley traction, and sometimes he had succeeded in getting the head in by pulley traction at the first attempt. In others he had been obliged to go further, and had been compelled to apply traction at intervals of a fortnight or three weeks or more. The subject was one the literature of which was enormous, and it was one of great interest, and he had no doubt it would bear much discussion.

Mr. Edmund Owen desired to join in congratulation to Mr. Collier and Mr. Fairbank on the results of their cases. When he was recently at St. Mary's Hospital, Mr. Collier told him he would like him to look at five cases of congenital displacement of the hip-joint which he had operated upon by a modification of Lorenz's method, and the modification was that alluded to by Mr. Tubby, making an incision over the obturator nerve in the pelvis and pinching that nerve. And as he had been immensely struck with the results shown him at St. Mary's, and Mr. Collier said he would be showing a further series at the Medical Society meeting, he made it his business to be present. He thought that pinching of the obturator nerve was going to do much in those cases, and perhaps it was; he certainly thought so when he came there. But when he saw Mr. Fairbank's cases he was equally astonished and pleased to find that Mr. Fairbank had obtained such perfect results, quite as good as Mr. Collier's, leaving nothing to be desired, and yet he did not pinch the obturator nerve. Therefore he would ask Mr. Collier and Mr. Fairbank to settle between themselves whether that pinching of the obturator nerve was an important feature in the treatment of congenital dislocation of the hip-joint. Mr. Collier said he believed there was less pain in the after-treatment if the obturator nerve had been pinched and the adductor muscles had been paralysed. He had been asking Mr. Fairbank if he thought there had been much pain in his cases, and he said that after the first few hours there was no particular pain and the children got used to the condition. He would like Mr. Collier and Mr. Fairbank to settle between them whether there was really any difference in the amount of pain which the child suffered under the two methods of treatment. There was no way in which one could judge of the amount of pain felt by a child. Mr. Collier said the pinching of the obturator nerve was followed by paralysis of the muscles. He would like to hear from Mr. Collier when that paralysis made its appearance, and how long the paralysis of the muscles was likely to last. The adductor magnus had a different nerve supply than from the obturator, namely the great sciatic, and he asked whether anything special had been done to that nerve, or had the treatment been directed simply to the obturator nerve?

Mr. Charters Symonds said his experience of that operation was not very great, but he had done it a few times, and he had been very much disappointed with his results. After what had been said in the discussion he was inclined to attribute it chiefly to the patients having been too old for it to be successful. Still, there lurked in his mind the idea that it was possible his work had been imperfect. He succeeded in getting the head of the bone into place. He would like to add his congratulations to the authors for the results they had obtained and for the demonstration they had given.

Mr. Collier, in reply, thanked the Society for the way in which they had spoken of his cases. With regard to the specific questions which had been put, several of them could not be answered at present, because neither Mr. Fairbank nor himself had made up their minds what was the perfect course. He thought what they would do in the future was to adopt the method of pinching the nerve in the younger children where there was

less likely to be any great tearing of the muscles. He was himself inclined to continue the method of pinching the nerve, and he thought Mr. Fairbank's cases were as successful as his own could be in his hands. Mr. Fairbank spent much time in the out-patient department looking after those children, more time than he himself seemed able to give to them. He maintained that the child which had its nerve pinched was less liable to get a return of the dislocation than a child was whose nerve was intact. With regard to the question of pain, careful observations would be made.

Mr. FAIRBANK, in reply, said he was very grateful to all who had spoken for the kind things which they had said about the cases. With regard to the obturator nerve, it might be of interest to say that he had seen that nerve injured in the ordinary operation of Lorenz more than once, so that all the muscles supplied by the obturator nerve remained paralysed, or at all events, weak for over a year after the operation. So that in the ordinary operation, without wanting to injure that nerve, it was sometimes paralysed.

CASES OF SPASMODIC TIC TREATED BY SYSTEMATISED EXERCISES.

Three cases of spasmodic tic treated by systematised exercises were described. All three cases belonged to the same class of disease—clonic coordinate muscular spasms more or less beyond the control of the will. A very strenuous effort of will could control the movements for a short time only. They were all of some duration. One of the patients, a boy, had had the attacks for ten years, the second had had them for fifteen years, and the third ten years. In the worst case, that of the boy, the movements came on three years ago after a fright, and they affected the respiratory muscles and the head and neck muscles, so that it was absolutely impossible for him to follow any occupation. The boy was put on respiratory exercises and arm movements in February, and he had had prolonged treatment during many hours of the day, at intervals, in the West End Hospital for Diseases of the Nervous System, but without any good effect at all. It could not be explained why there had been no effect upon him. In Case 2 the patient had had the same process of treatment. At one time her condition was worse than that of the boy now was—so bad that she could not sit upon a chair. She started exercises in February, 1907, and when she did them well she breathed inwards while expanding the arms. The great point was to get the patients to do the exercises slowly and at frequent intervals. She used to carry them out for five minutes every hour in the day, using the muscles chiefly involved. She had muscular twitchings referable to the face, the fits having started about ten years ago. About two years ago the movements spread to the neck, somewhat like spasmodic torticollis. The grimaces and twitchings of the head and neck were so great that she could not go to a public place. Last May she had started with systematic movements of the head and neck. It was a very frequent condition, and it was useful to know the kind of movements which did most good. The patients were instructed to follow with their eyes something on the ceiling and move the head with the eyes, taking the vision straightway down to the feet. This was done ten times on each occasion. The next movement was of the head from right to left. Then the patients were asked to do them diagonally from shoulder to shoulder very slowly and regularly, the idea being to re-educate the muscles. The exercises were given her to do, and finally she did them three times a day. The effect had been most happy, because after three months of those movements the condition had totally disappeared. In addition the patient had only had a mixture of gelsemium and bromide of arsenic. It must be remembered that the movements had been going on in that distressing way for something like ten years. At the People's Palace Gymnasium and Polytechnic Gymnasium numerous cases of spasmodic tic occurred among the pupils, and it was thought that if such patients were induced to bend their attention steadfastly on normal systematised movements of the affected muscles regularly and sufficiently often, the unstable paths and powers of inhibition would be re-educated. In nearly every case where the patient was able to give sufficient time and attention, cure resulted. The pathology of these spasmodic tics appeared to him to be fairly simple. Briefly, it consisted of an explosive motor impulse along a too well-beaten nerve-track. The principle on which the treatment depended was the replacement of those explosive morbid movements by slow, rhythmical, systematic, healthy movements of the same muscles at frequent

intervals during the day, the patient's attention being fixed steadfastly on these movements. Some cases were incurable, but in others recovery and improvement were very striking.

Dr. LEONARD GUTHRIE thought there were different periods in the course of the tics. There was one period in which the tics were produced by an uncontrollable impulse. The patient felt a strong desire to perform a certain action. He resisted the desire with all his power, but eventually he yielded, the patient performing those weird antics because of his uncontrollable desire to perform them. But as patients suffering from tics went on, he thought that the uncontrollable impulse seemed to vanish, and then the tic itself became a pure habit, more or less unconsciously performed. In the first case the tic was constantly performed, and done deliberately; the patient felt he could not help performing it, but later on, when the attack became habitual, there was no longer that uncontrollable impulse to perform it, and the patient did it more or less unconsciously. He asked in what period the exercises should be used—in the first, or in the second? His own feeling was that the exercises should only be used in such cases as he (Dr. Guthrie) mentioned last, namely, cases in which it was due to mere habit, and no longer due to uncontrollable impulse. In all cases of tic the condition was psychomotor, motor manifestations of a psychic unrest or instability or irritability. At the bottom of them all, in the early stages at all events, there was some condition of mental disturbance, and he thought that in the early stages, when they occurred, it was more important to regard the psychical side of the question than the motor. If one attended to the psychical disturbance, one could leave the motor manifestations to take care of themselves. He was now speaking more especially of children. Those who came into that condition were usually of a neurotic inheritance, and were themselves neurotic, they suffered from all kinds of emotional disturbance, and their proclivities to tics were largely due to their surroundings. In every case in which a child had what was called a tic, it was essential to regard the surroundings rather than the tics themselves. One had to consider the effect of nagging mothers, of overbearing sisters and brothers, of bullying schoolmistresses. Those were things which weighed on the mind of the neurotic child, and predisposed it to attacks. But there were other factors besides the surroundings; there were the exciting causes. He thought all tics originated in some minor irritation, which might be in the eye, or in the nose, or in the ear, or in the throat. He could remember a child was playing and had some dust in its eye, and that was followed by conjunctivitis. That caused much blinking, and the blinking went on for months longer than the irritation which caused it. There was nothing else wrong with the eyes. Another child sniffed when it had a cold, and the child was still sniffing although it had lost its cold. The point he wanted to convey was that the performance of the tic long outlasted its cause. And that raised the important question whether one should treat those minor irritations of eyes and ears and so on. He thought not, for the reason that the causes which produced them were very slight, and the tics went on long after the cause had disappeared. Therefore when a child blinked the proper treatment was, not to have its eyes examined, or when it sniffed to have its nose examined, but the proper treatment was to remedy the surroundings of the child, to take it away from all conditions which were likely to upset it, and leave the tics to take care of themselves. What he would like to conclude by saying was that as long as tics were produced by an uncontrollable impulse treatment by exercises was of little use, but when they became purely habitual he thought the exercises were of the greatest value.

A Case of Traumatic Epilepsy successfully treated by Operation.

Dr. F. S. PALMER said the case was extremely interesting, not only from the success of the surgical treatment, but because during the twenty-two years of his life he had twice been subjected to an operation for the relief of intra-cranial irritation. As the history was somewhat modified by what the patient told him, he would read the notes as corrected. They were to the effect that the patient came in on April 4th, 1908, complaining of severe headache and fits. The patient stated that ten years ago he fractured his skull; he believed a portion of a bridge fell upon him. In 1904 he received a second injury in the same region. The original blow was over the right parietal bone, and on the second occasion he fell off his bicycle,

injuring the same spot. In February, 1906, he was passed into the Army, and while on the march in India he suddenly became unconscious. Evidently he had an epileptic fit, and that was followed by several other attacks of a similar nature. On July 1st, 1907, he had three severe attacks, and became hemiplegic on the left side. The diagnosis was made that it was cerebral compression, and he trephined over the right Rolandic area on July 15th. The paralysis gradually disappeared, and by the end of December he could walk without difficulty. After the operation he had about sixteen fits in India, and in November, 1907, he was invalided home and sent to Netley. During the five weeks of his stay in Netley he had four attacks. At the end of the year 1907 he was discharged unfit for further service. Since then the fits had recurred at frequent intervals, and the headaches had become increasingly severe. His appearance in the out-patient room in April last year did not suggest the condition of ordinary epilepsy; he was intelligent, answered questions promptly, and no traces of his former hemiplegia could be found. His gait was normal and the movements of his limbs perfect. Both pupils were equal and reacted to light. Both eyes were normal, and there was nothing abnormal in his cranial nerves; the nervous reflex motor and sensory symptoms, except that the knee-jerks were a little exaggerated on the right side, were normal. Specific disease was denied. Frequently before the attacks commenced he had a definite sensory aura, which he described as a sensation of cold waves rolling over his left arm. Sometimes he could arrest an attack by tying a cord round his arm. Close to the cicatrix of a previous operation a depression could be felt. He was recommended for admission with a view to operation, but could not come into the hospital until December 15th. On January 7th he had a severe Jacksonian epileptic attack. The fit commenced with twitchings on the right side of the face and neck, and that was followed by clonic convulsions of the left arm and leg, becoming generalised. X-ray examination of the head showed a dark shadow near the cicatrix, which was very definite. He was taken in, and Mr. Ballance operated on February 17th. Mr. Ballance could not attend the meeting, but had sent the following report: "On reflecting the scalp flat over the seat of the previous operation two surfaces of depressed bone were observed, and these were obviously trephined circles of bone which had been depressed inwards after they had been replaced at the close of the former operation. A portion of skull 2 in. by 2½ in. was removed and the dura was adherent. There were two depressions in it corresponding to depressed circles of bone. A flap of the dura mater was made, and nothing abnormal was shown. The dural flap was sutured, and the bone removed and not replaced. The scalp flap was sutured, and dressing applied as usual. On May 26th the scalp flap was drawn down and a platinum plate fixed over the opening of the skull by a platinum wire. The patient had done exceedingly well since the operation, with no return of headaches and no recurrence of the fits."

Mr. WARREN LOW asked how long it had been since the last operation. He asked because he had congratulated himself on the result of a case three or four years ago, that of a man who came to St. Mary's Hospital who had had a bullet-wound in one of the battles of the Tugela, and there was a distinct depression in the skull at the junction of the occipital and parietal portions. Some months before he saw the man he had begun to have definite fits, and Dr. Harris and he saw him together and suggested that there was something wrong with the membrane underneath that point of the fracture. He found there a depression of skull, and the membranes were adherent at that point. A disc of bone was removed, and he did not open the dura mater, for it seemed normal, except for the adhesion. The patient did well for nine or ten months, and then his fits recurred. One had to be a little careful in congratulating oneself in a case of the kind until a considerable interval had elapsed.

Dr. PALMER replied that he could feel a distinct depression of bone over the site of the previous operation; that was confirmed by X-ray examination, which showed a dark shadow. He agreed with the remarks of Mr. Warren Low, but thought the circumstances in that particular case pointed to a hopeful result, because the pressure had been entirely removed. Some adherent dura was found, and the bone removed was not replaced, and the cerebral cortex which was exposed was found to be healthy. He agreed, however, that one could not be yet quite certain about it.

September 26th, 1910.

(*To be concluded.*)

SEVEREST ANÆMIAS.

'Severest Anæmias, their Infective Nature, Diagnosis, and Treatment,' is the title of an excellent work by Dr. William Hunter, published by Macmillan & Co. The title of the book indicates its scope and the general character of the anæmias with which it deals. For reasons which appear in every chapter and on almost every page, Dr. Hunter has definitely abandoned the use of the term "pernicious" for any form of anæmia, and has done so out of regard for the patient and in the interests of clinical medicine, because this term connotes nothing but confusion—historical, clinical, ætiological, pathological, diagnostic, prognostic, and hæmatological—without its equal in the history of any disease or group of diseases. Dr. Hunter has given the severe disease known to English observers as Addison's anæmia the position amongst infective diseases to which it is in his observation entitled, and the title "Addisonian," which connotes it and nothing else. The title "septic" has been selected as the most appropriate one for purposes of ætiological characterisation and treatment, to denote those anæmic conditions which most resemble the Addisonian anæmic disease and are most commonly mistaken for it. For the ætiological factor common to them all, which gives them their resemblance to each other and to Addisonian anæmia, is septic infection. The conditions dealt with in the book are very entangled in their history and obscure in their characters. Dr. Hunter has endeavoured at every stage by every possible means to make them clear to his readers. Wherever possible he has grouped the facts—historical, clinical, ætiological, pathological, or otherwise—in tabulated summaries, or represented them graphically by schemes, maps, charts, and plates. The history of their observation and interpretation is dealt with in full detail, and a full bibliography, extending to some 400 papers, is appended up to the year 1903, when this work was commenced. The standpoint from which the whole subject of these severest anæmias is approached is ætiological, clinical, and pathological. He is satisfied that their infective and clinical characterisations go far deeper into their real nature and carry further in their diagnosis and treatment than those which have regard solely to the more purely hæmatological criteria, which observers have continuously striven to connect with these anæmias. Dr. Hunter has endeavoured to bring out this fact in every chapter of this work, for he is satisfied that its proper appreciation is of essential importance in the early diagnosis and effective treatment of these anæmias. The results here recorded break down once and for all the misleading ætiological and diagnostic traditions associated with, and largely created by, the ill-chosen title, "pernicious." They substitute for these traditions the new facts with old facts in new relations, which, according to Dr. Hunter's observations and interpretation during the past twenty years, afford a fuller and clearer conception of the nature of these anæmias and furnish surer guides for their early recognition and effective treatment. The anæmias here dealt with are very common. If recognised in their early stages—as they definitely can be by their infective characterisations here described, but not by their blood characters—they can be arrested. This applies even to Addisonian anæmia. Dr. Hunter's sustained interest in this anæmia is due to the fact that in endeavouring to lay bare the ætiological foundations of this, the most mysterious and severe of all blood diseases, he has found himself digging deep among the foundations of all severe anæmias. The result has revealed that the ætiological factors which give them their severity are of infective nature, and that the seat of this infection in by far the largest proportion of cases is the alimentary tract. This outcome is of no mere pathological interest. On the contrary, it is of the utmost practical importance. Both classes of facts afford indications in the treatment of these diseases which are daily yielding better and better results, and are destined as they become more fully recognised to remove from most of these anæmias the characters which have hitherto given them their severity. The attention of the profession is invited more especially to the singular history of the original and erroneous identification of Addisonian anæmia with Biermer's "progressive pernicious anæmia" to the character of the cases termed "pernicious," on which Prof. Ehrlich made his first blood observations, and to the singularly misleading ætiological and hæmatological traditions which, in consequence of these observations, have for the past thirty to forty years gathered around the unhappy and unfortunate name of "pernicious" anæmia. Dr. Hunter considers that so far as Addisonian anæmia is concerned, the whole facts of the disease place this malady among the group of infective diseases as firmly and securely as the pathological lesions, clinical features, and clinical course of scarlet fever place it among recognised infective diseases.

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CONTENTS.

PAGE

*A Lecture on Abscess and Fistula. By F. SWINFORD EDWARDS, F.R.C.S. ... 401

*A Lecture on Meningococcal Meningitis. By J. HUGH THURSFIELD, M.D., F.R.C.P. 407

*Demonstration of Cases at the Medical Society of London 412

Rheumatism 416

* *Specially reported for the Clinical Journal. Revised by the Author.*

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The Index and Title-page for Vol. XXXVI will be published with the next issue of THE CLINICAL JOURNAL.

A LECTURE

ON

ABSCESS AND FISTULA.

Delivered at St. Mark's Hospital.

By F. SWINFORD EDWARDS, F.R.C.S.,

Senior Surgeon to the Hospital.

GENTLEMEN,—This being the second lecture on abscess and fistula, I think it would be well to recapitulate a little of what I said on the last occasion. You will recollect that, speaking of rectal abscess, I said that it may originate either outside the bowel—from traumatism, septic infection, as in gonorrhœal prostatitis or in periostitis—or it may be due to an ulcer or abrasion of the mucous membrane through which fæcal matter passes into the submucous tissue. I am of opinion that in the large majority of cases the fistula is due to an infective process proceeding from an ulcer or abrasion in the mucous membrane of the anal canal. Hence, in the majority of cases one would expect to find an internal opening, and that in point of fact is the case. It may not be very apparent, but the experienced surgeon will often succeed in discovering the internal opening where others less skilled have failed. Through this opening a foreign body, such as a fish-bone, may pass into the submucous tissue, and on opening the abscess you may find the foreign body there. The four situations in which I showed an abscess in connection with the lower end of the rectum may occur I have put down here as follows: First there is the submucous abscess; secondly, the subcutaneous abscess. The submucous abscess is divided from the rectum by mucous membrane, and is between the mucous and muscular coats. The next most common is the ischio-rectal abscess, which is deeper than the subcutaneous, and is only limited in its deep aspect by the levator ani muscle. The fourth variety is far less common: it is an

abscess in the pelvi-rectal space, which space is above the levator ani and between that muscle and the bowel. I pointed out that this is generally due to some prostatic trouble—usually suppuration in connection with gonorrhœal infection. Most of these abscesses result in fistulæ. The only way to prevent such a result is by a thorough and prompt evacuation of its contents. After freely opening the abscess by a T-shaped incision, pass in the finger and break down all loculi, temporarily pack with gauze or wool, and then apply fomentations. This is the best way to obviate the chance of a fistula resulting. We now come to the varieties of fistula, viz. the complete fistula, which has its external orifice in the skin outside the anus and its internal orifice in the mucous membrane of the bowel and usually between the two sphincters. Here is a diagram of a blind internal fistula—that is to say, a fistula having only one orifice, and that in the bowel. This is a blind external fistula, having one orifice in the skin, the sinus running up towards the bowel. The blind internal fistula may burrow downwards, as it usually does, towards the skin before it finds its way through and becomes a complete fistula. A blind internal fistula may become a complete internal fistula by opening again into the bowel, thus having its lower and upper orifice in the mucous membrane itself. A complete external fistula is not unknown—that is to say, the fistula may have its internal orifice in the anus, the track running below the external sphincter. Then I described the varieties of complete fistula. The most noteworthy of these is the **horseshoe** form. I pointed out that this fistula is usually due to an abscess forming behind the anus. It is prevented coming immediately to the surface owing to the thickness of the tissues in the middle line posteriorly, due to the ano-coccygeal ligament and the insertion of the levatores ani and to the sphincter ani, which comes forward from the tip of the coccyx. All these structures make it difficult for abscess to find its way to the surface in the middle line. It tends, therefore, to burrow on either side, and will often open about half-way between the posterior and anterior limits of the anus. This abscess is due to the fact that an abrasion has occurred in the anal canal situated midway between the two sphincters. The resulting abscess burrows on either side of the anal canal in the way I show you in the diagram, so forming a fistula shaped like the Greek letter Ɛ. When you see the buttocks of an individual burrowed and riddled as if it were rabbit warren it is probably due to a horseshoe fistula. A **semi-horseshoe fistula** is one in which this takes place only on one side. From the external orifice the track runs backwards to the mid-line behind the anus and then runs forwards to the internal orifice, which is always situated in the mid-line dorsally. Then I passed on, in the last lecture, to describe how the internal orifice bears a certain relationship to the external orifice, and for the operating surgeon this is very important, because when he has before him a case of fistula he ought to know, or be able to make a shrewd guess, from the position of the external orifice, where the internal orifice will be. If you draw a line transversely through the centre of the anus, and you find the external orifice is behind this imaginary line, you may be almost certain the internal orifice will not be immediately opposite it, but you will find it in the mid-line dorsally, and therefore you will have a semi-horseshoe or curved fistula to deal with. It does not matter where your external orifice is so long as it is behind this transverse line; it will take a curved course and open into the bowel, passing through the dorsal aspect of the rectum. But if the external orifice is in front of this plane you will find the internal orifice immediately opposite it. Perhaps the only exception to this rule is when the internal orifice is situated in the mid-line anteriorly. Here the external orifice is usually to one or the other side.

As to the operations necessary for the cure of these conditions, supposing the surgeon sees the external orifice and he has not recognised that he is dealing with a semi-horseshoe fistula, having inserted the director he may feel its point under the mucous membrane, possibly close to the real internal orifice, and he may think that his probe-pointed director is lying in the track of the fistula, push it through the mucous membrane, and may slit it up. I have several times seen scars of this kind coming to the hospital, where the fistula has not been laid open at all. In fact the patient still has his fistula intact, but with a scar showing what had been done. Of course, in operating for fistula, the object to be aimed at is to lay open the track, and nothing but the track. You should first of all pass your finger into the bowel, palpate carefully with the thumb of the same hand, trying to

approximate it to your finger all round, and thus ascertain the course of the indurated track. In a semi-horseshoe fistula it will turn backwards, and you will probably recognise the internal orifice situated in the mid-line dorsally. Now pass in the probe-pointed director and slit up to some point behind the anus. Now change the direction of the director, and pass it from the extremity of this incision forwards through the internal orifice and slit up, thus dividing the external sphincter in the mid-line dorsally. You have thus only divided the sphincter in one place, and that in the best manner—that is, at right angles to its fibres. The same proceeding may be employed for complete horseshoe fistula, though it is often possible to tackle it in another way. Let us imagine that you recognise you are dealing with a complete horse-shoe fistula. You have two or more orifices behind the transverse line already referred to. Slit up these tracks, making for a point posterior to the anus. The resulting wound may be extensive and deep, notably the transverse incision, which has laid open the sinus connecting the tracks on either side. In such a case it is well to be content with this, and to leave the bowel untouched for the present. Allow the patient a month or so for the wounds to partly heal. Then complete the operation by passing a probe-pointed director from the centre of the transverse wound through the internal opening—in other words, you will simply have to slit up what is now an ordinary straight dorsal fistula, which will take possibly a fortnight or three weeks to heal. This operation in two stages has much to recommend it, indeed, I have known such a fistula to close without the second operation being necessary. Another point is that by this method you are dealing with a wound which is extra-rectal, and can be kept free from all fæcal contamination.

Concerning the operation for blind internal fistula, you must remember that this variety is often overlooked, as is the case with the horse-shoe and the semi-horseshoe fistulæ. The patient complains of some discomfort about the anus He now and then notices a drop or two of pus, but there is nothing to be seen outside. You may pass your finger into the bowel and not detect the small internal opening. Now proceed to palpate with your other hand, if you like, or with the thumb of the same hand. You will find an area of induration either at some depth from the surface, or it may be immediately underneath the skin. The matter is then a very simple one. Lay open the induration as you would an abscess by incising with a sharp-pointed bistoury, then pass your probe-pointed director through the wound into the bowel and complete the operation in the usual way. But if the fistula tracks upwards you may not feel an induration outside, in which case it will be necessary to expose the internal orifice and pass a director from within outwards. The best speculum for this purpose is the duck-bill. If your probe-pointed director runs upwards under the mucous membrane, there is no reason why the tract should not be laid open, but you must supplement it by forcible dilatation of the anus, otherwise the drainage will be inefficient, indeed, you may even divide the sphincter, as the effects of forcible dilatation do not last long. I have often maintained forcible dilatation for some minutes, and on examining the patient two, three, or four days afterwards, I have found the sphincter as tight as ever. Supposing you have before you a case of multiple fistulæ, one complete and straight, and another a semi-horseshoe one, absolutely distinct, what would you do? Would you operate on both fistulæ at the same time and possibly divide the sphincter in two places? It might be done, but I consider it to be risky. I should, in such a case, operate on the worst of the two fistulæ, in this case the semi-horseshoe one, laying it open in the usual way; thus the sphincter is set at rest by dividing it dorsally. The other fistula should be curetted and afterwards treated by some injection, such as nitrate of silver twenty grains to the ounce, once or twice a week; in this way we should expect it to heal. If it did not, I should not hesitate to lay it open in the ordinary way after the first wound was healed. It is better to divide the external sphincter in two places, bearing in mind the possibility of incontinence following, than it is to divide both the internal and external sphincter once. In case of fistula I practically never divide the internal sphincter, except its lowest fibres. When a fistula has its internal orifice in the *pars analis*, let us say an inch from the verge of the anus, the internal orifice is felt by the finger as a depression, but when higher up in the bowel it is usually nipple-like in shape and stands out above the level

of the surrounding mucous membrane. It is somewhat hard and indurated, and is due to a mass of granulations. The relation a high-lying sinus bears to the internal sphincter is an important one. When lying between the muscular and mucous coats, lay it open with the rest of the fistula by all means, but if it is situated outside the muscular coat it should not be slit up on account of the risk of incontinence after division of both sphincters. If it runs up outside the internal sphincter, leave it alone and trust to curettage and nitrate of silver injections.

With regard to **incontinence**, supposing a patient has been operated upon perhaps more than once for fistula, and incontinence has occurred, that is to say, that the patient cannot retain either flatus or motion. In the milder cases there is only incontinence of flatus, but still that is distressing. What can you do for the relief of this condition? The milder forms of incontinence are very often to be remedied by linear cauterisation of the lower part of the mucous membrane of the rectum, cutting as you withdraw your knife well into the external sphincter, so as to produce cicatricial contraction; this is done in four lines, anterior and posterior, and one on each side. But if you are dealing with a serious case where there is incontinence of fæces you must do some plastic operation. Here is an anus which is gaping. You pass your finger in and ask the patient to prevent himself having an action. You will find the sphincter is very weak, and that there is no contraction on your finger at all. The best thing to do is to make a curved incision on both sides of the anus, and deepen that incision, keeping outside the sphincter, until the wound is an inch in depth. Then take hold of the centre of each margin with forceps and pull them apart. You will have made a diamond-shaped wound. Now pass your sutures—generally four are required—in such a manner that the anterior extremity of the wound is approximated to the posterior. On tying the sutures the line of union will radiate from the anus instead of being parallel with it. Having repeated this on the opposite side the anus will be much contracted, as shown here, and it may be quite difficult to insert the finger. For this to be successful you should obtain union by first intention. In order to ensure this it is important to keep the parts as aseptic as possible. Before operation the skin is thoroughly sterilised, and be sure the bowel is properly emptied. It should be sterilised by washing out with an antiseptic lotion, for which I find lysol good. The region of the anus should be shaved and thoroughly washed with ether soap and subsequently with sublimate solution. The operation having been done there is only one further step, and that is to keep the part from being contaminated with flatus, for these patients are kept at first upon a slop diet, which readily produces flatus. Therefore in these cases I always pass in a small india-rubber tube the size of one's little finger, about three or four inches in length, and let it project well from the anus, so as to carry away any flatus which may be formed. I also put on a collodion dressing, thus trying to seal it hermetically. Other layers of gauze are placed above this, the tube projecting beyond them. This, I think, gives the best chance of healing by first intention. Subsequently the bowels, which have been confined for four days, should be opened by giving something to produce a fluid motion. I find a powder of two grains of calomel combined with two grains of pulv. elaterii co. most useful. This will generally produce, at least a soft, if not a fluid action. At the same time I instruct the nurse to give an olive-oil injection through the tube (which you remember was inserted at the operation) before taking it out. This is very important, because the patient then passes the motion without the pain and straining which might otherwise be associated with it.

There is the question of the preparation of patients for operation of fistula, and also the after-treatment. With regard to preparing the patient, if I am operating on Monday morning at 9 o'clock I like the patient to come into the hospital or the Home at least two days before, and on the Friday night or early Saturday morning an ounce of castor oil is given. Early on the Sunday morning another dose of castor oil is given, but rather a smaller dose, viz. from half an ounce to six drachms. On the evening of Sunday the bowel is washed out with a warm boracic enema, and on the morning of the operation, about 6 o'clock, another warm boracic enema is given. That is the usual method of preparing patients for operations on the lower bowel, be it a case of fistula, hæmorrhoids, fissure, polypus, or what not.

With regard to the operation for fistula, it is well

to be provided with three or four probe-pointed directors, the points of which are of different sizes. Usually I have three sizes—small, medium and large. That is very important. You may ask, What is the good of a large one when a fine one will do all the work? A fine one is necessary, because sometimes the track will only admit the finest probe-pointed director. Will not that do also when you have a large fistulous track to deal with? No, because the tissues about the fistula are soft and easily perforated, and a very fine director is apt to be pushed outside the fistulous tract into the cellular tissue. Take the case of an ordinary straight fistula. It is laid open, and then each side is clipped with a compression forceps and held apart, so that you may see the track which you have divided. It should show up well, lined with granulation-tissue often of pure colour, indeed, darker in colour than the rest of the wound. If there is any doubt, I employ a Volkmann's spoon and scrape off the granulations. If there is a branch leading off the main track its whereabouts will be shown by granulations which the spoon cannot remove. Insert the probe-pointed director at this point, find where it goes, and lay it open. The next thing to do is to render the wound as superficial as possible. For this I employ Salmon's back cut. A knife is drawn along the floor of the track coming out well through the skin, thus freeing the part which had been bound down. I then remove all overhanging edges of the skin. Often the mucous membrane abutting on the wound in the bowel falls into it and may delay the healing process. If so it should be treated as a pile, and removed by cutting and ligation. This ensures the fistula being laid open in the best way, and you then have a gutter left without overhanging edges of skin. All bleeding is now stopped; but there is, as a rule, very little bleeding. A vessel or two may have to be tied. The wound is now packed with sterilised cotton-wool, which has been soaked in some antiseptic, such as carbolic acid or sublimate solution, and squeezed nearly dry. A narrow strip of this is laid along the fistula, and on the top of it I put some more swabs which have been wrung out in sublimate solution. Over this place a pad of cotton wool and a T-bandage. This dressing is left undisturbed until the following day, when the external swabs are changed. The internal dressing is left unchanged until the first action of the bowels. If it becomes offensive and loose before that it should be changed. In extensive cases hot boracic fomentations are commenced the following day, and changed every four hours to loosen the dressing with which it has been packed, or the patient can sit in a hot boracic bath, which is comforting, and which, I think, promotes healing. Hereafter the wound is *lightly* dressed, *not plugged* with lint or gauze, or things of that sort, but lightly dressed with absorbent cotton-wool, which has been wrung out nearly dry in sublimate solution. I have several times operated upon patients who have had an operation done by other surgeons, and they have been distressed to learn that they have had to undergo another operation for fistula. I have asked them what it is they dread, and they reply that it is the after-dressing that is so painful, and that they cannot adequately express all they went through on the last occasion owing to the pain. I have seen men dress fistula cases, and I can understand why it is that patients are hurt. The only way to avoid hurting your patient is by inserting the index finger of your left hand, well coated with vaseline, into the bowel in order to open the part and to direct the insertion of the dressing. I have seen men take a strip of lint or gauze and a director and at once ram it along the wound into the bowel. Could anything be more likely to cause keen pain? I place the patient on the side and ask him to strain down, anoint my finger with vaseline and pass it well into the bowel, and having my dressing ready and a rod without any sharp point, I pass it up alongside my finger into the bowel, placing it gently into the fistula. You do not want it to act as a foreign body, but to prevent adhesion between the two edges. I do not like to hear doctors say they have *plugged* a case every day, because it means you give a patient pain every day, and probably delay healing. In the case of a complete fistula which only passes through the lower fibres of the external sphincter you may think it will be enough to lay open that fistula, but it is not. You must supplement it by making an incision through the base of your wound, cutting through a few more fibres of the external sphincter, or you may find it necessary to cut through the whole of the sphincter. If you prefer not to do that, then, having cut through some of the superficial fibres of the sphincter, practice forcible dilatation so as to put the part at

rest, otherwise healing will be slow and unsatisfactory.

An extensive operation for horseshoe fistula is one from which it might take a patient two or three months to get well, whereas some of these smaller fistulæ take only ten days or a fortnight to heal. In operating for fistula do not forget to keep to the track and to see the track as you go on with your operation. If you do not understand the "geography" of the part find your way step by step, always keeping to the track. If the sphincter has not been divided this should be done, or forcible dilatation carried out.

I should like to say a word about the condition known as **sinus over the sacrum and coccyx** and **fistula in tubercular subjects**. With regard to sinuses over the sacrum and coccyx, every now and then you will find in the skin covering these bones an external opening, and a probe will pass towards the bone. You may look upon that as an external orifice of a fistula *in ano*, and you think it must communicate with the bowel. Or, again, you may think this is a sinus leading to diseased bone, and proceed to lay the bone bare with the view of removing a sequestrum; but in so doing more harm than good will result. A sinus over the sacrum and coccyx generally originates from abscess owing to a blow or fall, just as fistula *in ano* often results. These conditions are practically never due to disease of bone. I once saw a case which a colleague had thought was a sinus leading down to necrosed bone in the sacrum or coccyx. He cut away a good deal of the bone but he never cured his patient, simply because he was not conversant with the lesion with which he was dealing. These sinuses are often branched, and there may be several openings. The floor of them is formed by the fibrous tissue at the posterior aspect of the sacrum and coccyx, and all you have to do is to lay them well open, curette, and pack. Do not expose any bone in your operation, and do not try to force the director towards the bowel, or you will be doing the patient irreparable harm, for you are attempting to deal with a condition which really does not exist.

The next question is, Do I advocate operation on fistula in phthisical cases? That depends on the relative condition of the lung and the local lesion. Supposing the patient to have acute phthisis and to be suffering but little pain from the anal fistula, I should not recommend any cutting operation. Supposing the patient to have active phthisis and also to be suffering a great deal of pain on defæcation—because very often these cases have a large internal orifice, and at every motion of the bowels fæcal matter will pass into that fistula and cause considerable pain, and in some cases agony—in such cases the sphincter must be divided so as to give the patient rest. If you are dealing with a case of arrested phthisis, with acute rectal symptoms, you will, of course, operate at once. Where the phthisis is quiescent and there is a small fistula, with a small internal orifice not causing any discomfort to the patient, I should be loth to operate. I should send the patient into a suitable environment and prepare him for operation, and if I did operate I would limit the division of the sphincter as much as possible, for when you completely divide the sphincter in phthisical cases incontinence may result. The difference between the appearance of a healthy fistula and a tubercular one is fairly well marked. In the tubercular case the skin over the fistula is boggy, and bluish in colour, and the orifice is undermined and appears as a distinct hole, whereas in a healthy, long-standing case you will have a button-like projection marking the site of the external opening, and leading from this towards the bowel you will feel an indurated track, often described as resembling the stem of a tobacco-pipe.

October 3rd, 1910.

Dr. J. P. DeWitt considers that the fact that present methods of treatment will effect a cure in tuberculosis in 90 to 95 per cent. of cases if applied early should stimulate all medical men to become competent to make an early diagnosis. One should proceed to the use of tuberculin to prove the diagnosis. The inunction of the tuberculin ointment of Moro or the cutaneous test introduced by von Pirquet is usually employed first; if reactions do not occur, the old tuberculin of Koch is injected subcutaneously. After gradually increasing the dose from one-tenth to ten milligrammes, if no reaction has occurred it is quite certain that the patient has not early tuberculosis. Few patients in limited financial condition ever recover from well-developed tuberculosis. Observation will show that many supposed to have been cured by rest and feeding relapse. He has never seen harmful results from reactions, but often marked improvement follows where previously the patient was not doing well. Patients immunised with tuberculin do not develop complications of the larynx, glands, or bowels, and very few relapses are noted, while patients apparently cured without tuberculin frequently relapse and develop complications.—*Journ. A. M. A.*, vol. lv, No. 3.

A LECTURE

ON

MENINGOCOCCAL MENINGITIS.

Delivered at the Hospital for Sick Children, Great Ormond Street, W.C.

By J. HUGH THURSFIELD, M.D., F.R.C.P.,
Assistant Physician to the Hospital.

GENTLEMEN,—If you look into the text-books of medicine which were published ten years ago you will find in most of them a description of a disease called chronic basilar meningitis. It was first described in this country by Gee and Barlow in 1878, under the title of "Cervical Opisthotonos in Infants." Since then it has had several names, but in England it is probably best known as posterior basic meningitis.

I have called your attention to these dates because the history of the advance of our knowledge of this disease is a striking illustration in medicine of that progress which is by many supposed to be confined to the realm of surgery. In thirty years we have learnt much regarding the causes, prophylaxis, diagnosis, and treatment of a disease which then was merely a mystery. This afternoon I propose to review briefly the present state of knowledge, and indicate the many problems which still await solution.

History.—There has for a long time been known an epidemic disease called epidemic cerebro-spinal meningitis, never very common, but occurring from time to time in all civilised communities. In 1887 Weichselbaum, of Vienna, described the result of his researches into the bacteriology of the disease, and his results have been confirmed many times. The disease is due to an intra-cellular diplococcus, which in its morphology resembles closely the gonococcus, but differs widely from it when cultivated on laboratory media. In 1898, Dr. Still, who was at that time Registrar and Pathologist at the Hospital for Sick Children, was able to establish that the disease known as posterior basic meningitis was constantly associated with the presence in the meningeal effusion of an intracellular diplococcus, which agreed in most respects with the organism described by Weichselbaum. The question whether these two organisms are identical is by some bacteriologists regarded as still uncertain, but I think I am justified in stating that the prevalent opinion is that the two are identical, and as a corollary, that posterior basic meningitis is the sporadic form of epidemic cerebro-spinal meningitis. This conclusion is, moreover, strongly supported by the clinical evidence which is now available owing to our greater certainty of diagnosis.

Morbid anatomy.—I am going to assume that you are familiar with the appearances of the meninges seen at the post-mortem examination—the thick creamy effusion limited to the base of the brain, and to a well-defined area, that comprised between the anterior boundaries of the circle of Willis and the under surface of the cerebellum and medulla and pons. Occasionally the effusion spreads along to the superior surface of the cerebellum, and appears also in two small areas at the tip of each temporo-sphenoidal lobe. In addition, it occupies the whole of the posterior surface of the cord. In cases of any standing the ventricles of the brain are dilated, and in many of those patients who recover from the original attack a chronic hydrocephalus supervenes and destroys life in some months. Apart from the changes in the cerebro-spinal system, the rule holds that no other disease is present in the body. I must add that though these appearances are characteristic of the disease, they are also found at times in meningeal infections with other organisms—for instance, the pneumococcus.

Clinical symptoms — I propose to describe briefly the chief features of the disease clinically, and afterwards to illustrate from actual cases the ways in which these are grouped.

Onset.—The invasion of the disease varies in character to an extraordinary degree. In some cases it deserves the name "*fulminant.*" A girl, æt. 15 years, was in her usual health all day, but in the evening complained of a slight headache; she went into another room, and was found there a few minutes later prostrate, semi-conscious, and moaning with pain in her head and limbs. She quickly developed all the signs of a meningitis, and after a severe attack recovered. A man of forty years or more similarly complained of feeling out of sorts, went to bed, and next morning was unconscious, delirious, and within twenty-four hours of the first malaise was covered from head to foot with a pur-

puric rash ; he was dead within forty-eight hours of the first symptoms.

Such cases are fortunately rare in the sporadic form of the disease, though, to judge from the accounts of the epidemics, they there form an appreciable percentage of the whole number.

The more common form of onset is the *sudden* attack of fever and vomiting, mild at first but increasing rapidly, and soon complicated by retraction of the head, squints, rigidity of the limbs and convulsions. In infants under two years of age the first symptom is almost invariably either vomiting or convulsions, the vomiting being often almost continuous, but the convulsions, as a general rule, repeated, if at all, at long intervals. Lastly, there is the *insidious* onset, met with very often in infants, very seldom in adults, when the first symptom noted is a slight stiffness of the muscles of the neck, with screaming when the head is moved, and often when the limbs are straightened ; or again, the child may be brought because the anterior fontanelle is bulging, and it appears to be wasting and restless. The majority of the cases which it has been my chance to observe have fallen into this last category, but I have an impression that in the last year or eighteen months I have seen an acute onset occur rather more frequently than I did two or three years ago. This is, however, a mere impression, and even if it were established as a fact it might be due to purely lortuitous circumstances.

Symptoms.—The cardinal symptom of the disease is *retraction of the head*, or, as it was originally described by Dr. Gee, cervical opisthotonos. This is a symptom which I believe I may say is invariably present. It is, however, not necessarily present, either in the early stages or at any stage, in a marked degree. In the greater number of instances, however, it occurs within the first few days of the onset, and is sufficiently marked to attract immediate attention. The head may be merely tilted so that the chin is directed slightly upwards, or it may be drawn so far back that the occiput rests upon the spine between the scapulæ. In the less marked cases it is usually possible to place the head in the normal position, though only at the cost of considerable pain to the patient; when the retraction is extreme the rigidity of the muscles is so great that the child can be lifted by the head without altering its position in relation to the body. In the more acute cases the retraction of the head frequently is intermittent, and may in a few days disappear entirely, so that it is possible that by the time the patient comes under observation that this particular symptom is no longer available. The extreme degree of cervical opisthotonos is rarely seen in any other disease, but some retraction of the head is not infrequent in the late stages of tuberculous meningitis, and may occur in children in whom there is no trace of meningitis at all.

Vomiting in the acute cases is almost constant at the onset, but may afterwards become infrequent ; in the chronic cases it is of daily occurrence, with, however, remissions from time to time. The vomiting is, as a rule, cerebral in its character, and occurs without any definite relation to feeding. It is closely associated with the progressive emaciation, which is such a distinctive feature of this form of meningitis, as compared with tuberculous meningitis.

Rigidity of the limbs again varies much. In the extreme cases the arms especially are so contorted that their position would *à priori* seem to be impossible ; the whole arm is pronated to an extreme degree, the wrists and fingers firmly flexed ; the leg stiffly extended, with the feet almost in a line with the tibiæ, and the toes curled up under the foot.

In the less marked cases the rigidity, though present, often requires to be elicited by examination. One method of such examination is that known as Kernig's sign. This sign, which has, in my own opinion, obtained an undue importance, is best elicited by placing the patient at full length on his back, and then seizing the toes and lifting the foot until the leg and thigh form a right angle with the pelvis. In nearly all cases of this form of meningitis the hamstring muscles are so rigid and contracted that long before the right angle is reached the limb is violently flexed at the knee-joint. The value of this sign, however, as an indication of the presence of meningitis is largely discounted by the fact that it can be obtained in a considerable number of patients suffering from diseases that have no connection with any lesion of the meninges. It should not, however, be neglected, since when the proper discount is allowed it remains a valuable confirmatory sign.

Fever.—In all the acute cases there is con-

siderable fever, and it is probable that even in those instances where the onset is insidious and where there is no fever at the time they come under observation, there has been some rise of temperature earlier. The character of the fever varies very considerably, as will be apparent from the charts I show. Occasionally it is a continued fever with a slow decline, resembling a chart of enteric fever; again, it is from the beginning an intermittent fever; or again, a fever wholly irregular with remittent and intermittent curves. Sometimes the fever drops by lysis; at other times it falls suddenly by a crisis. With the fever there is usually a marked rise in the pulse-rate, but comparatively slight disturbance of respiration.

The *eye-symptoms* are important. Strabismus is fairly common, but not, I think, nearly so often seen as in tuberculous meningitis; in young infants lateral nystagmus is frequent, and the pupil reflexes are nearly always sluggish. But the characteristic sign is the wide-open eye, the staring upward gaze—in marked contrast with the photophobia seen in tuberculous meningitis. The palpebral fissure is wider than normal, and very often a ring of sclerotic is seen above the cornea. I do not know what the physiological explanation of this peculiar symptom may be, but it is very often seen in association with a temporary blindness. It is, of course, in young children always difficult to ascertain whether there is or not good visual acuity, and especially so in children who are the subjects of acute illness; but the frequency with which blindness is met with in older children makes it fairly clear that its occurrence in young children is not over-estimated. It is found in at least 50 per cent. of all cases that survive more than a fortnight. So far as my own experience goes, if recovery takes place the eyesight invariably recovers also, but there are, I believe, cases on record of permanent blindness. As to the cause of this blindness there is no sufficient explanation; but it is quite clear that it does not depend upon any inflammation or choking of the optic disc. Throughout the disease, except in the rarest instances, the optic disc remains clear and free from swelling. In some cases it seems even to be a paler white colour than is usual, suggesting an early stage of atrophy. In a recent acute case, however, though there was no papillitis, the optic discs were certainly suffused, and showed no appreciable difference from the surrounding retina; in this instance, however, there was no blindness, though there seemed to be a diminution of visual acuity.

But though the optic nerve escapes permanent damage, it appears to be quite certain that the patients are not always so fortunate with regard to their auditory nerves. I am told that there are not a few deaf people in whom the origin of the deafness can be traced to an attack of this form of meningitis. I am not aware that I have ever recognised this symptom in posterior basic meningitis, and in the cases that I have seen recover from the attack it has certainly not been present. It is, however, said to be a not uncommon complication in epidemics, and to be followed invariably by complete deafness.

The *skin* is often the seat of eruption. In the more acute attacks purpuric eruptions are fairly frequent, and in the fulminant cases are almost constant. The purpuric maculæ cover the whole surface of the body and limbs, and have given rise to the name "spotted fever." In the chronic cases such purpuric eruptions are very rare, and, except in one instance, the most I have ever seen was a small number of petechiæ on the legs and abdomen. Herpes of the lips and face, and occasionally in other situations, is much more common, occurring early in the disease. It does not appear to bear any relation to the severity of the attack. Other skin eruptions are macular erythemata, resembling that of measles, and an urticarial eruption about the face, neck, and shoulders. Hyperæsthesia of the skin is not infrequent; it is often apparently general in distribution, but may be strictly localised; anæsthesia is also described, especially in the epidemic forms.

The patellar reflex is usually active, but is seldom exaggerated, while the plantar reflex is, so far as I have been able to obtain it, normal. The abdominal skin-reflexes, on the other hand, appear to be abolished as a general rule.

A peculiarity of this disease is the involvement of the joints; this usually occurs when the disease is fully established, and if recovery takes place the joints also recover quickly. The knee-joint seems to be that most often affected, and the organisms have been many times obtained from such swellings. In at least one case, however, the swelling of the knee-joint preceded any symptoms of meningitis, and I have also seen one instance in which an

organism apparently identical with the *Diplococcus intra-cellularis* was obtained from a swollen knee-joint in a patient who was not otherwise ill. It would seem, therefore, that in uncomplicated synovitis of obscure origin this organism is a possible cause.

The *sequelæ* of the disease are, with two exceptions, unimportant. The first of these I have already mentioned, *i. e.* the occurrence of permanent deafness; if this should occur in a young child before the age of talking, deaf-mutism is the inevitable result, but, as I have said before, though from written accounts it would seem to be not infrequent in epidemic, it is certainly very rare in the sporadic form of the disease. The other result is chronic hydrocephalus, and this is unfortunately by no means rare. In the majority of such cases the hydrocephalus is slowly but steadily progressive, and causes a fatal termination to the disease some weeks or months after the acute stage is passed. In rare instances the hydrocephalus appears to remain stationary at a stage when the child may yet retain sufficient cerebral substance to enable it to be educated.

Diagnosis.—In the diagnosis of the disease the first step is to establish the existence of a meningitis, and in the majority of cases the history of the onset with convulsions or vomiting, the strabismus, the rigidity of the limbs, the fever, and delirium, or coma renders this fairly easy. Yet there are difficult cases. In young infants it sometimes is very difficult to establish the diagnosis as between meningitis and chronic wasting disease, and this is especially the case when the onset is of an insidious character. In such infants there is the lethargy, the emaciation, the vomiting, and in some cases the rigidity of the muscles and the retraction of the head. In these doubtful cases the course of the disease will usually clear up the diagnosis in a few days, even without the aid of lumbar puncture. This proceeding is the chief aid in the diagnosis, not only of the presence of meningitis, but of its nature. In nearly all cases of meningococcus meningitis, at any rate in the first fortnight, the fluid obtained by lumbar puncture is turbid to the naked eye, sometimes even purulent. When examined by the microscope the turbidity is found to be due to the presence of numerous polymorphonuclear leucocytes, in which it is usually easy to demonstrate the characteristic Gram-negative intra-cellular diplococcus. In tuberculous meningitis, on the other hand, the cerebro-spinal fluid is usually clear, contains very few cells, and those chiefly lymphocytes. Moreover, when placed upon a suitable medium and incubated, in the great majority of cases of meningococcic meningitis an abundant growth of the organism takes place in twenty-four hours, whereas in tuberculous meningitis the media remain sterile. In the absence of the assistance afforded by this means of diagnosis the distinction between tuberculous and diplococcic meningitis is not always easy. Before the employment of lumbar puncture as a routine mistakes were often revealed upon the post-mortem room table. But the three points on which I should lay stress are: (1) The age of the patient. Posterior basic meningitis is common under one year of age; tuberculous meningitis, though it occurs at that age, is far more often met with after the age of three years. (2) The character of the eye-symptoms. In posterior basic meningitis the eyes are wide open, do not close even in a bright light, and often exhibit the sclerotic above the cornea; in tuberculous meningitis, on the other hand, there is nearly always photophobia. (3) Rigidity of the cervical muscles with retraction of the head is an early symptom in posterior basic meningitis; it is a late symptom, if it occurs at all, in tuberculous meningitis.

In older children, and in the more acute attacks, the difficulty at first is to decide whether the meningeal symptoms are indications of meningitis or whether they are the early symptoms of a pneumonia, typhoid fever, or some other acute infection In this difficulty the examination of the cerebro-spinal fluid is of the greatest importance, and next to this the estimation of the number and character of the patient's leucocytes. In both tuberculous and meningococcic meningitis the polymorphonuclear leucocytes are, as a rule, slightly increased. though as a rule more in the latter than in the former disease, while in pneumonia there is a marked leucocytosis, and in typhoid fever a marked leucopenia.

There are from time to time instances of cerebro-spinal meningitis described which are not due to the *Diplococcus intra-cellularis.* These are rare, though of great interest from the pathological and bacteriological point of view. One such epidemic was carefully investigated by Dr. Gordon, and

shown to be due, not to the *Diplococcus intracellularis*, but to the *Micrococcus catarrhalis*, and in other cases the *Bacillus influenzæ*, or at least an organism culturally and morphologically indistinguishable, has been found.

Treatment.—The researches of bacteriology in the disease seemed for some time to be of no practical value in the treatment of the disease, but in the last five years there has been a great stride made in the treatment by means of serum-therapy. There are to-day several serums which have been used with a varying degree of success, both in the acute epidemic and in the sporadic cases. The process of manufacture of these differs in detail, but in principle is the same. Of the serums which have been used I will briefly mention three—Ruppel's, Wassermann's, and Flexner's. Of the first alone have I had practical experience. It is made by taking a pure culture of the meningococcus and growing it in a liquid medium, the precise nature of which is not stated by Ruppel. After several days' growth on this medium, however, the organism acquires an enhanced virulence, so that a subcutaneous inoculation of small quantities is quickly fatal to rabbits and guinea-pigs. Ruppel claims that by immunising such animals by means of minute doses gradually increased he has been able to produce a high degree of immunity. At this stage Ruppel began to immunise horses, and has produced a serum which is capable of conferring on animals a high degree of immunity, provided the serum is inoculated early in the course of the infection. He finds, for instance, that if the serum and the coccus are inoculated together the serum is completely protective, whereas every day's delay increases the mortality, and that after the fourth day the serum is practically useless.

I have used Ruppel's serum in several cases. In one, that of a child, æt. 2½ years, the effect was remarkable: within twenty-four hours of the subcutaneous inoculation of the serum the fever fell by crisis, and the child made an uninterrupted recovery. The attack was acute, and the change which took place in the patient's condition made a great impression on me. Another case was that of a girl, æt. 14 years, who had an acute attack. She had an injection beneath the skin on the tenth day of the disease, and although there was a slight improvement in her condition on the following day, there was no further improvement until four days later, when there was again exhibited a sudden crisis with a remarkable disappearance of all symptoms. A third case in some ways is the most noteworthy. A child, æt. 2 years, was admitted in the third week of the disease with all the characteristic symptoms; it was very seriously ill and I gave a bad prognosis, but ordered that lumbar puncture should be performed and Ruppel's serum inoculated beneath the skin. A marked improvement followed immediately; the lumbar puncture was repeated four times, at intervals of four or five days, and a second injection of the serum was given a week after the first. The child showed marked improvement, and though progress was slow, eventually recovered. In other cases the results were less obviously good, and in the chronic cases I have had no success.

It will be obvious that no conclusions can possibly be drawn as to the value of the serum from these records, more especially as the experience of other observers is in direct contradiction, but personally I feel that in one case the result may fairly be attributed to the serum, and am encouraged to give it a further trial.

Wassermann's serum I have not used, but his last report on the subject shows encouraging results in the treatment of epidemic cerebro-spinal meningitis; he also finds that the earlier the serum is administered the lower the mortality, and adds that it is apparently useless in subacute or chronic cases. Dr. Robb, of Belfast, reported in February, 1909, on the use of Flexner's serum in the Belfast epidemic. This serum is prepared by the immunisation of horses, and is given direct into the spinal canal after removal of the cerebro-spinal fluid. The success of the treatment is marked; Dr. Robb gives a careful account, and from his figures is, I believe, justified in claiming that the use of this method has lowered the mortality from 80 per cent. to 25 per cent.

The results, therefore, of serum treatment are remarkably promising, and personally in future I intend to treat all the cases which come under my care with one or other of these serums. Ruppel's at present is easily available and has yielded what appear to me to be good results, but if I find on further trial that it is not so useful as it appears to be I shall use Flexner's. There are other serums sold, but of them I know nothing.

Next in value to serum-treatment I should place lumbar puncture, repeated at frequent intervals; it is a little operation easily performed, especially in children, and if care is taken to remove the needle directly there is any indication of collapse there is no danger. In some instances repeated puncture in itself has appeared greatly to benefit the patient, but in most cases I admit that it seems to me to be without any great therapeutic value.

Cases have also been treated by the withdrawal of the cerebro-spinal fluid and its immediate injection beneath the patient's skin; the theory is that the subcutaneous tissue is an unfavourable medium for the development of the coccus, and therefore a favourable medium for the development of immunity. I have no experience of the method. Successful cases have also been recorded by the inoculation of a vaccine made from the organism cultivated from the patient. I have tried this method in three cases, but without any beneficial effect, so far as I could observe, though one patient was said to have improved for a time.

Apart from these methods of treatment there does not appear to be any means of attacking the cause of the disease, and it only remains to place the patient in the best conditions and treat *secundum artem* any symptoms which appear. In epidemics the mortality, if this course be adopted, is very high indeed, and does not appear to be any lower in hospitals amid healthy surroundings than in the houses of the poor. In the more chronic cases which occur sporadically the mortality is still very high, but does appear to be influenced by the better surroundings of the hospital.

There are several interesting points on which I have not had time to touch, for instance, the question of prophylaxis, with which is intimately concerned the problem of ætiology. It has been hitherto supposed that the path of infection is by the nasal mucous membrane to the base of the brain, but there are considerable difficulties in the acceptance of this view, and the occurrence of the micrococcus in the nasal discharges is now known not to be so common as was formerly believed. A theory which has received some support is that the primary infection is intestinal, and that the organism attacks the lumbar cord and thence makes its way upwards to the base of the brain. The evidence in favour of this is, however, at present quite insufficient, and the whole question must remain an open one.

October 3rd, 1910.

DEMONSTRATION OF CASES AT THE MEDICAL SOCIETY OF LONDON.

(*Concluded from p.* 399.)

A Case of Total Laryngectomy.

Mr. H. Clayton Greene showed a case of total laryngectomy on which he was asked to operate. There was an intrinsic growth of the larynx, which was found to be endothelioma. The dangers of the operation were fully explained to the patient, and she consented to have it done. He made a median incision, turning flaps from either side, and the larynx was removed. And, as usual in such cases, he found the operation was only half completed; the growth had spread to the pyriform fossa, the posterior wall of the pharynx, and under the end of the œsophagus. Therefore he had to abandon the operation in an uncompleted state, or to go on to remove the whole pharynx, posterior as well as lateral walls, and the vertebral columns and upper end of the œsophagus. The patient's condition was so good that, with the advice of those who were helping him, he did that, and there was no trouble attending it, and no bleeding. The œsophageal tube was sewn into the upper end of the œsophagus to prevent leakage, and the opening, which had been divided, was packed with the tube. The rest of the wound was lightly sutured, and was packed with carbolic gauze. He attributed the fact that there were so few complications in the way of sepsis to the fact that it was Kocher's method in removing the tongue. But Kocher's operation is not properly described; that which is described as such leaves out the important point in it that he packs the wound with carbolic gauze and keeps it packed. They put the patient in a reclining position, with the head low, so that any matter would tend to trickle away from the mediastinum. There were no complications of any kind except what was to be seen now, namely, a small fistulous track above the tracheotomy wound, which is no doubt due to the ligature. The ablation of the pharynx made it necessary to provide an artificial passage, and that was done by a Symond's tube. The points which impressed him in the case were that endothelioma tended to remain local rather than to become a generalised disease, and there were no signs of dissemination in the glands. Secondly, that in ordinary cases it was important

to give the patient the hope of the chance afforded by an operation. Since the operation the patient had had comparative comfort, she had put on weight, and could lead an active life, except that in the cold weather she suffered from bronchial catarrh. But she had no complaints to make about her present condition. She could make herself understood, and the operation, although very severe, had been justified by the result. It was easy to feel the Symonds' tube when the tracheotomy tube was removed.

A Case of Osteitis Deformans.

Dr. Essex Wynter and Mr. T. H. Kellock showed a case of osteitis deformans. The patient was a female, æt. 42 years, married. She had always been healthy formerly. Three brothers died in infancy; four sisters were alive and well. The patient had had one still-born child only. Eight years ago she began to suffer pain in the ankles and legs after standing. Four years ago she first noticed deformity of the left leg. The general nutrition was good. The left femur was much thickened and bowed forwards, the right similarly affected but to a less degree. The tibiæ were somewhat concave on the internal surface, but did not appear to be thickened. There was slight lordosis, and the space between the costal margins and crests of the ilii was diminished. The bones of the skull did not show any marked change. There was one inch shortening in the left leg as compared with the right. The bicipital ridges on the humerus were exaggerated on both sides. The patient lost the right hand as the result of an accident when a girl. A skiagram of the left femur showed the bone to be enlarged in all dimensions, and the shadow cast by it to be abnormally dark. The urine was normal.

Mr. Warren Low said that Mr. Kellock only referred to the relationship between malignant disease and osteitis deformans, and he (Mr. Low) had that brought to his notice the other day in which the other side of the question was presented, namely a patient came with malignant disease of the cæcum, involving the ilium, from which he shortly afterwards died. He was then found to have osteitis deformans, and probably he had had it for some years. He did not know whether Mr. Kellock had found out what that relationship meant, but there could be no doubt that it existed. On the other hand he had had a case of osteitis deformans who certainly had had the disease ten or fifteen years, and did not appear to be much the worse for it. There were enlargement of tibia and femur, and a large mass on one humerus, and that was how it was discovered because he had broken the other humerus, and some years afterwards that humerus was examined and a lump was found on the right humerus, whereas on the left humerus it had been broken.

Mr. Kellock, in reply, said that the association between the two diseases was well recognised, in fact the last female patient whom he had seen with osteitis deformans—and he took it that the condition was rather uncommon in them—had a sarcoma of the lower end of the femur, and had marked osteitis deformans in the rest of her body. The tumour was removed and found to be periosteal sarcoma. He believed that the majority of sarcomata arising in osteitis deformans had been medullary ones, but in the present case it was periosteal. She died of recurrence in the lungs or some other viscera, and she had for many years been the subject of osteitis deformans. He hoped the present patient was not promising to do the same thing.

Case of Abdominal Hydatids.

Mr. Warren Low showed a case of abdominal hydatids, which was shown before the Society by Dr. Bertrand Dawson in November, 1903. Mr. Low said he brought it forward to complete the report of the case. In 1903 it was brought forward as a case for diagnosis. The patient originally developed a tumour in his abdomen in Pretoria—he was an ex-R.A.M.C. officer—in 1900, and by a curious coincidence Mr. Low saw him in Africa, when the condition first started. He was shown at the Society as a case for diagnosis, and the general feeling which members expressed then was that it was hydatids. He was operated upon at the London Hospital by Mr. Eve, who found hundreds of cysts, some of them large ones, filling the abdominal cavity. There were none in the liver. He removed as many as he could, but could not do a radical operation. The man was in hospital four months, and when he left he said he was much relieved by the operation. But he came back six years afterwards with his abdomen in the condition which was seen that evening,

obviously full of cysts, in some of which the characteristic thrill could be felt, and also, as Mr. Kellock had that evening pointed out, a curious sound was heard which he (Mr. Low) had not previously been aware of, when one percussed and used the stethoscope, like a tight drum or tambourine would emit. There was a large mass in the left side of the abdomen, which one could easily feel *per rectum*. They were obviously filling up the pelvis, and there was no doubt some pressure on the bladder as he had some urinary disturbance, and he came to the hospital complaining of pain in the anterior crural nerve. He (Mr. Low) had no doubt it was from pressure of the large mass. If any Fellow had seen a similar case he would like to ask what was the best thing to do for the man. Any operation which might be performed could only be partial: only a small portion of the cysts could be removed. Would drainage of a large cyst relieve him, or had any member knowledge of any other method of treatment which might be useful?

Mr. KELLOCK said he thought Mr. Low had a big task in operating on that patient. He (Mr. Kellock) saw the patient some years ago, when it was a question of what the diagnosis was. But anything in the shape of operation seemed to be rather negatived, except such an operation as opening the abdomen and tapping each individual cyst. And then one could see the surrounding spaces, so that the fluid should not disseminate over the peritoneum. He would be inclined to try that if the case were under his care without trying to take away very much. Mr. Low had just mentioned what was a very interesting point about hydatids. It was brought to his (Mr. Kellock's) knowledge many years ago by accident. He was listening over an abdomen containing hydatids and somebody percussed it close to his stethoscope, and it produced a most remarkable noise. He believed anyone who had once heard that noise through the stethoscope would never forget it. It resembled the sound produced by a tight tambourine or drum more than anything else. He supposed it was to be explained by the vibration of the tense wall of the cyst. It was very important that one should tap some of the hydatids which one had the stethoscope on. If one did not get both fingers on the same cyst one did not procure the hydatid thrill, and if one gave the cyst a sharp tap close to the stethoscope one obtained that extraordinary note.

Mr. MALCOLM said he met a case like that some years ago, and he attempted to operate, but did too much, and the patient died. He then made up his mind that if he saw another case of the kind he would attempt to remove as many of the cysts as he could easily get at. His impression was that it might be relieved in that way, and that by taking away and doing many operations a cure might possibly be obtained. He did not think the hydatids did any harm except by pressure, and if a lot of them were removed the others would get more room and the patient might get better for a time. In the report of the case when shown before, it was said that something must be done, and the condition was now so bad that something must be done if he was to be saved. He had had other cases also which he had operated upon, but none of them were exactly like the present one, as the present patient had them all over the abdomen.

Dr. WEST asked whether there was any evidence to show where the hydatid originated. He remembered a case of the kind where there had been a cyst in the liver, which was tapped, and evidently it must have leaked. It was a proliferating cyst, and they must have sown themselves into the peritoneum and grown there. Ultimately the case became septic, and the patient died. With regard to tapping, he always used to teach that hydatids in the liver might be safely tapped, but if the condition was outside the liver ~~the~~ condition might become septic. And that he still believed to be true. He thought there would be an objection to tapping any of those cysts when they were lying free in the intestines, that they would almost certainly become septic, and then the patient would die, as did the one he mentioned. The hydatids perforated into the vagina from the bladder, and the whole thing rapidly ended. Therefore he would say that a large operation repeated several times would be safer if anything was to be done, and evidently something must be done.

CEREBRAL TUMOUR.

Dr. J. WALTER CARR showed a doubtful case of cerebral tumour. He said he brought the patient for diagnosis. It was clear that there was present some serious intra-cranial mischief, but he felt doubtful about what the nature of it might be. That there was an increase of intra-cranial pressure

was evident, and he believed the boy had become worse during the last few days. If he had thought he would get worse, as he had done, he (Dr. Carr) would have done lumbar puncture. There was increasing apathy, difficulty in swallowing, and some loss of control of the sphincters, as well as paralysis of both sixth nerves, accounting for the convergent strabismus, due, not to localised pressure, but to the general intra-cranial pressure affecting the sixth nerve before the other oculo-motor nerves. One could dismiss ear trouble or abscess in connection with it, because there was no indication of any cause for such abscess. He thought it most likely that the boy had a tumour. As to where the tumour was it was difficult to say, but it was more likely to be in the cerebellum than anywhere. Naturally in a child one thought of the cerebellum first of all, and because as time went on he had marked difficulty in walking, and the characteristic gait associated with a cerebellar lesion. But the patient could not walk or even stand now. What the nature of the tumour might be he could not say. It was thought possibly it might be tuberculous, but the Calmette reaction was negative. Another possibility was that the boy had chronic meningitis, causing hydrocephalus. But that case did not conform to the meningitis which he was familiar with, and he was not familiar with chronic meningitis at that age. He could not remember having come across a case in a child of six. The history would support that idea. It was three months since the boy began to have cerebral symptoms, and the symptoms at first were slight, chiefly changes in position, and during the last week they had become more marked. There was irritability of temper, and then the development of strabismus, and he thought the meningitis would have caused more rapid development of symptoms. The boy had not had hydrocephalus and head-retraction. Neither had he had vomiting. But whether he had had tumour or meningitis, there was complete absence up to the present of optic neuritis, of headache, and of vomiting. During the last month he had been sick once, and that was only very slightly, and followed the taking of too much food.

Dr. ENGLAND said there was in hospital the case of a man, æt. 54 years, five or six weeks ago. That particular patient had most of the typical symptoms in connection with cerebellar tumour—reeling gait and vomiting—and died in one of the attacks of vomiting; he believed it was from asphyxia or syncope. A post-mortem examination was made but nothing abnormal could be found anywhere in the cerebellum. But there was one little patch of suspicious pia mater which it was thought might be tuberculous, but the specimen was sent for examination and report, and no definite reply that it was organically wrong had been received.

RHEUMATOID ARTHRITIS.

Dr. F. S. LANGMEAD showed a case of rheumatoid arthritis in a boy. He said it might be regarded by some as an instance of Still's disease. He did not think such a condition should have a special name; it was only rheumatoid arthritis modified in character by the youth of the patient. The mother of the patient was subject to myxœdema, and there was a good deal of evidence showing that changes in the thyroid gland had some relation to rheumatoid arthritis. Cases of rheumatoid arthritis had often paroxysmal tachycardia. In Dr. Still's cases he noted exophthalmos in two out of twelve. Exophthalmia had occasionally been described in other cases of rheumatoid arthritis, and enlargement of the thyroid gland had often been described in association with rheumatoid arthritis, and all the signs of Graves' disease had been found present in the condition. Therefore he (Dr. Langmead) considered that the relationship of thyroid gland to rheumatoid arthritis was a question well worthy of consideration, and that was why he showed the case.

October 3rd, 1910.

FOR the cure of a recurrent tumour Janeway resected a portion of the ulna five and a half inches long, which ran through the centre of the growth. A fragment of bone of the same length, and three eighths of an inch wide and one eighth of an inch thick, was chiselled off from the crest of the tibia, together with its adherent periosteum. This was then placed between the two extremities of the ulna remaining and fastened in place with silver wire. The periosteum of the implanted fragment was also sutured to the periosteum of the remaining portions of the ulna. The wound was closed, with the exception of a small drain at each end. In two months' time the patient could freely use the arm.—*Journ. A. M. A.*, vol. lv, No. 10.

RHEUMATISM.

THE causation of rheumatic fever and of the chronic affections of the joints generally described as rheumatic is not generally known. Many observers have found micro-organisms in the blood and synovial fluid of patients suffering from acute rheumatism. A very large number of different bacteria have been described in this connection—staphylococci, streptococci, diplococci, and bacilli. For practical purposes the question of the microbial origin of the disease is at present associated with the claims of an organism first discovered by Apert and Triboulet (1898), and subsequently investigated by Wassermann, and in this country by Poynton and Paine. Walker calls the organism the *Micrococcus rheumaticus*. It would be preferable, perhaps, to call it a streptococcus, as it appears to belong to this group. With regard to the specific nature of this organism, no certain verdict has been pronounced as yet. The opinions of some authorities as to the unity of the streptococci have already been mentioned. On the other hand, it is difficult to believe that so distinct a clinical disease as rheumatism can be due to the same cocci which produce suppuration. If the rheumatic cocci be merely an attenuated form of these, it is curious that suppuration is practically never seen in rheumatic joints, as it might have been expected that the organisms would not infrequently gain increased virulence in susceptible persons. There is no reason to believe that rheumatic subjects are in any way refractory to suppurative lesions. Walker, however, finds that the rheumatic cocci will grow in a filtered culture-fluid in which other streptococci have been grown and have died out. This, which has been alluded to as Marmorek's test for the unity of the streptococci, appears to show that the *Streptococcus rheumaticus* is distinct from ordinary pyogenic organisms, whilst many observers consider that the organism isolated from the joints is a variety of the pneumococcus. It must be mentioned that some observers have failed altogether to find micro-organisms in cases of acute rheumatism. Thus McCrae examined 270 cases with practically negative results, and Triboulet, finding organisms in some cases and not in others, considers that the sterile cases represent the true disease, while those due to organisms only simulate rheumatism. The serum used by Menzer is prepared from streptococci derived from human sources. The effects produced in cases of chronic rheumatism are very remarkable, as, according to Menzer's account, the injection of the serum gives rise to a reaction of an inflammatory nature at the seats of rheumatic lesions (the joints), which is followed by subsequent improvement. This peculiar result of the serum would suggest, as was pointed out by Blumenthal, that the serum is not antitoxic or anti-bacterial, but contains a toxin similar to tuberculin or mallein. The reaction is exactly parallel to that which follows an injection of the old tuberculin in a patient suffering from a tuberculous joint. Menzer, however, holds that his serum is anti-bacterial, and from what is known of its mode of preparation it should be of this nature. He suggests the explanation that the reaction is due to destruction of the cocci present in the patient and to the resulting rapid absorption of intra-cellular toxins. Symptoms of constitutional disturbance also accompany the injections of serum in rheumatic patients, viz. rise of temperature, headache, sore throat, and enlargement of lymphatic glands. Cardiac disease, if present, may at first be aggravated by the remedy, but is subsequently improved. The local inflammatory symptoms are not seen in cases of gonorrhœal rheumatism which are treated with the serum.

The above is taken from a most interesting and instructive book on serums, vaccines, and toxins, by Dr. William Cecil Bosanquet and Dr. J. W. H. Eyre, published by Cassell & Co. The authors discuss the increased attention recently paid to tuberculin as a remedy for tubercular disease of all kinds, and observe that a more suitable scale of doses having been devised, the dangers inherent in the old scheme of treatment are thus practically banished. Under the heading of local use of serums it is advised in some diseases characterised by distinct local lesions to inject the serum in the neighbourhood of these in order to procure a local effect. Thus, in erysipelas good results have been obtained by injections of anti-streptococcic serum into the periphery of the inflamed area, and in plague it has been recommended to make the injection into some part of the skin which is drained by the lymphatics leading to the bubo. An anti-bacterial serum has been used in the form of lozenges in cases of diphtheria, in addition to the use of antitoxin subcutaneously. Toxins and antitoxins are fully discussed, and a unit-dose of diphtherial toxin is defined as that amount of any preparation of diphtherial poison which will just suffice to kill a guinea-pig weighing 250 grm. in a period of four days. This is also known as m.l.d., or the minimal lethal dose.

INDEX TO VOLUME XXXVI.

PAGE

Abdomen, palpation of the 246
Abdominal breathing, importance of, in case of weakness and insufficiency of the heart 118
,, hydatids, case of . . . 413
,, muscles, fibrositis of, by G. R. Murray 49
,, surgery, interesting cases of, by H. Curtis . . . 22, 42
Abscess and fistula, by F. Swinford Edwards. 401
Air-passages, foreign bodies in, by C. Wall . 161
Anæmia, simple infantile, by E. Cautley . 325
,, splenic, chronic malignant endocarditis simulating 73
Anæsthetics, two common errors in the administration of, by J. D. Mortimer . . 63
Analgesia, spinal 352
Andrews, H. Russell, some practical points in the management of difficult labour . . 251
Aortic murmurs 120
Aphasia and other disorders of speech, by T. A. Williams 268
Appendicitis and pregnancy, by W. H. Battle 313
Arthritis: its varieties and treatment, by W. Murrell 337
,, deformans, by O. Grünbaum . . 289
,, melitensis 341
,, rheumatoid 415
Ascites, chronic, with cirrhosis of the liver, by W. P. Herringham 65
Asthma, by A. F. Hertz 198
,, by H. Batty Shaw 86

Battle, W. H., in the wards of St. Thomas's Hospital 312
Bosanquet, W. C., some considerations on pleurisy 213
Breast infection, treatment of . . . 304
Bronchiolectasis 79
Bulbar paralysis, acute (polio-encephalitis inferior), in girl, æt. 13 years, by L. Guthrie 385
Bunch, J. L., skin rashes and their treatment 257

PAGE

Buzzard, E. Farquhar, early diagnosis of subacute combined degeneration of the spinal cord 374

Campbell, H., medicine on the continent . 53
Camphoric acid in treatment of sweats in tuberculosis 72
Cancer, two historical cases of, and the treatment of the disease, by F. M. Sandwith 8
,, of rectum treated by the abdomino-perinæal method, by H. J. Waring. 193
Catarrhal colitis, by H. P. Hawkins . . 34
Cautley, E., simple infantile anæmia . . 325
Cerebral tumour 414
,, ,, case of, by J. A. Ormerod . 343
Cirrhosis of the liver, with chronic ascites, by W. P. Herringham 65
Clinical demonstration at the Polyclinic, by Purves Stewart 353
Coal-tar derivatives *versus* opiates . . . 80
Colitis, muco-membranous, by A. F. Hertz . 263
,, treatment of, by H. P. Hawkins . 33
Colon, diseases of the, by W. J. Hadley . 337
Constipation, chronic, and its management, Lane's conception of 352
Continent, medicine on the, by H. Campbell 53
Corner, E. M., treatment in septic conditions 151
Cumston, C. G., diagnosis and differential diagnosis of pathological processes causing enlargement of the kidney. 377
Curtis, H., interesting cases of abdominal surgery, including some rare forms of intestinal obstruction 22, 42

Degeneration, subacute combined, of the spinal cord, early diagnosis of, by E. F. Buzzard 374
Dislocation, congenital, of hip-joint after Lorenz's operation 395
Dislocation, congenital, of hip, treated by a modification of Lorenz's operation . . 394

PAGE
Dunn, P., so-called second sight—its features, varieties, and causes 122
Duodenum, ulcer of, the non-surgical treatment of, by G. Herschell 273
Dupuytren's contraction, by W. H. Battle . 316
Dysenteric arthritis 341

Edwards, F. S., abscess and fistula . . 401
Emphysema, surgical, of abdominal wall, by W. H. Battle 313
Endocarditis, chronic, malignant, simulating splenic anæmia 73
Endosteal sarcoma of tibia, by H. J. Waring . 17
Endotheliomata of base of tongue, by F. Eve 113
Epilepsy, traumatic, successfully treated by operation 398
Epithelioma of pudenda, by W. H. Battle . 315
Epsom College 96
Eve, F., three rare tumours of base of tongue (two of them endotheliomata), with remarks on surgery of malignant disease of tongue . 113
Eye, on some pitfalls in diagnosis of common diseases of, by A. H. Thompson . . 286
Eye-muscles, on some affections of, by A. H. Thompson 29

Fats, some effects of over-feeding with, in certain cases of insanity 342
Femoral hernia, radical cure of, by C. B. Lockwood 321
,, ruptures and their management, by C. B. Lockwood 209
Fibrositis of the abdominal muscles, by G. R. Murray 49
Fistula and abscess, by F. S. Edwards . . 401
,, rectal, by F. C. Wallis . . . 369
Foreign bodies in the air-passages, by C. Wall. 161

Gall-stone diabetes 91
Gland, suppurating, by W. H. Battle . . 316
Gonococcic arthritis 338
Gouty arthritis or acute gout 338
Grünbaum, O., arthritis deformans . . 289
Guthrie, L. G., on acute bulbar paralysis (polio-encephalitis inferior) in a girl . . 385

Hadley, W. J., on diseases of the colon . . 337
Hawkins, H. P., treatment of colitis . . 33
PAGE
Heart, lectures on, by S. West . 97, 118, 137
,, weakness and insufficiency of, importance of abdominal breathing in case of 118
Hemiplegia and its treatment, by G. Holmes 129, 156
Hernia, femoral, radical cure of, by C. B. Lockwood 321
Hernial protrusion, penetrating wound of a (self-inflicted), by H. Curtis . . . 24
Herringham, W. P., chronic ascites with cirrhosis of the liver 65
Herschell, G., the non-surgical treatment of ulcer of the duodenum 273
Hertz, A. F., on asthma 198
,, on muco-membranous colitis . 263
Hip, congenital dislocation of, treated by a modification of Lorenz's operation . . 394
Hip-joint, congenital dislocation of, after Lorenz's operation 395
Holmes, G., on hemiplegia and its treatment 129, 156
Hutchison, R., on hysteria in childhood . 305
Hydatids, abdominal 413
Hyperæmic treatment of inflammation, by A. V. Poyser 139
Hysteria in childhood, by R. Hutchison . 305

Iliac constriction in intestinal stasis, by W. Arbuthnot Lane 16
Indigestion, treatment of, by E. I. Spriggs . 173
Infantile anæmia, simple, by E. Cautley . 325
Inflammation, hyperæmic treatment of, by A. V. Poyser 139
Inflammatory and septic conditions, use of vaccines in 328
Influenza bacillus in the respiratory tract . 220
Influenzal arthritis 341
Inguinal ruptures which are difficult to manage, and their treatment, by C. B. Lockwood 177
Inoculation of smallpox, the practice of, by F. M. Sandwith 317
Insanity, some effects of over-feeding with fats in certain cases of 342
Intestinal obstruction, some rare forms of, by H. Curtis 22, 42
,, stasis, iliac constriction in, by W. Arbuthnot Lane. . . . 16

Jenner, Edward, the life and work of, by F. M. Sandwith 328

PAGE

Kidney, enlargement of, diagnosis and differential diagnosis of the pathological processes causing, by C. G. Cumston . . 377

Labour, difficult, some practical points in the management of, by H. R. Andrews . . 251
Lane, W. Arbuthnot, iliac constriction in intestinal stasis 16
Laryngectomy, total 412
Lett, Hugh, on retention of urine . . . 281
Lewers, A. H. N., case of pan-hysterectomy for carcinoma of body of uterus, in which an infiltrated portion of bladder was removed 6
Liver, cirrhosis of, with chronic ascites, by W. P. Herringham 65
Lockwood, C. B., inguinal ruptures which are difficult to manage, and their treatment . . 177
,, on femoral ruptures . . 209
,, on radical cure of femoral hernia 321
Lorenz's operation, congenital dislocation of hip-joint after . . 395
,, ,, congenital dislocation of hip treated by a modification of . . . 394
Lupus erythematosus, by G. Pernet . . 221

Malignant disease of tongue, remarks on surgery of, by F. Eve 113
Medicine on the continent, by H. Campbell . 53
Meningitis, meningococcal, by J. H. Thursfield 407
Migraine, by G. Rankin 247
Mitral murmurs 122
Mortimer, J. D., on two common errors in the administration of anæsthetics . . . 63
Muco-membranous colitis, by A. F. Hertz . 263
Murmurs, heart 120
Murray, G. R., fibrositis of abdominal muscles 49
Murrell, W., arthritis: its varieties and treatment 337
Myomectomy during pregnancy, case of, by F. E. Taylor 47

Nephrotomy by the silver-wire method . . 86
Neuralgia, trigeminal, treatment of . . 101

Ophthalmia, sympathetic, by A. Ormond . 70
Opiates, coal-tar derivatives *versus* . . . 80
Ormerod, J. A., on a case of cerebral tumour 343

PAGE

Ormond, A., sympathetic ophthalmia . . 70
Osteitis deformans 413

Paralysis, acute bulbar (polio-encephalitis inferior), by L. G. Guthrie . . 385
,, agitans, case of 353
,, of arm, treatment of . . . 262
Pernet, G., on lupus erythematosus . . 221
Pituitary extract and some of its effects, by L. Williams 92
Pleurisy, some considerations on, by W. C. Bosanquet 213
Pneumococcic arthritis 340
Pneumonia, discussion on 357
Poyser, A. V., hyperæmic treatment of inflammation 139
Pregnancy, case of myomectomy during, by F. E. Taylor 47
Proctitis and sigmoiditis, by F. C. Wallis . 241

Rankin, G., migraine 247
Rashes, skin, and their treatment, by J. L. Bunch 257
Rectal fistula, by F. C. Wallis . . . 369
Rectum, carcinoma of, treated by the abdomino-perinæal method, by H. J. Waring . 193
,, innocent tumours of, by C. G. Watson 389
Retention of urine, by Hugh Lett . . . 281
Reviews and notices of books:
Abdominal Hysterectomy, by J. Bland-Sutton 198
Anæmias, Severest, by Dr. W. Hunter . 400
Serums, Vaccines, and Toxins, by Dr. W. C. Bosanquet and Dr. J. W. H. Eyre 416
Walker's Loose-leaf Pocket-book (Medical) 224
Rheumatic arthritis 337
Rheumatoid arthritis 338, 415
Riviere, C., tuberculous cases . . . 302
Ruptures, femoral, by C. B. Lockwood . . 209
,, inguinal, which are difficult to manage, by C. B. Lockwood . 177

Sandwith, F. M., smallpox and its early history 294
,, the life and work of Edward Jenner 328
,, the practice of inoculation of smallpox 317
,, the value of vaccination . 346
,, cancer and the treatment of the disease . . . 8

PAGE

Sarcoma, endosteal, of the tibia, by H. J. Waring 17
Second sight, so-called—its features, varieties, and causes, by P. Dunn 122
Septic arthritis, acute 341
„ conditions, treatment in, by E. M. Corner 151
Serum arthritis: malade sérum . . . 342
Shaw, H. Batty, on asthma 86
Sigmoiditis and proctitis, by F. C. Wallis . 241
Skin rashes and their treatment, by J. L. Bunch 257
Smallpox and its early history, by F. M. Sandwith 294
„ inoculation of, by F. M. Sandwith . 317
Spasmodic tic treated by systematised exercises 397
Speech, disorders of, by Tom A. Williams . 268
Spinal analgesia 352
„ cord, early diagnosis of subacute combined degeneration of, by E. F. Buzzard 374
Splenic anæmia, chronic malignant endocarditis simulating 73
Spriggs, E. I., treatment of indigestion . . 173
Stasis, intestinal, iliac constriction in, by W. Arbuthnot Lane 16
Stewart, Purves, clinical demonstration at the Polyclinic 353
Surgery, on some fundamental truths in, by C. B. Lockwood 1
Sweats in tuberculosis, camphoric acid in . 72
Sympathetic ophthalmia, by A. Ormond . 70
Syphilis, case of, by W. Hale White . . 145
„ discussion on 181, 205
Syphilitic arthritis 339
Syphilitic disease, case of 354

Tabes, case of 355
Taylor, F. E., case of myomectomy during pregnancy 47
Thompson, A. Hugh, on some affections of the eye-muscles . 29
„ on some pitfalls in the diagnosis of common diseases of the eye . 286
Thursfield, J. H., meningococcal meningitis . 407

PAGE

Tibia, endosteal sarcoma of, by H. J. Waring 17
Tic, spasmodic, treated by systematised exercises 397
Tongue, rare tumours of base of, by F. Eve . 113
Traumatic epilepsy successfully treated by operation 398
Tricuspid and pulmonary murmur . . . 122
Trigeminal neuralgia, treatment of . . 101
Tubercular disease, by F. C. Wallis . . 81
Tuberculin for treatment and diagnosis of tuberculosis 302
„ localised tuberculosis treated with, by Clive Riviere . . . 302
Tuberculosis, localised, treated with tuberculin, by Clive Riviere . . . 302
„ sweats in, camphoric acid in treatment of . . . 72
Tuberculous arthritis 340
„ sore in neck, by W. H. Battle . 312
Tumour, cerebral 414
„ „ case of, by J. A. Ormerod . 343
Tumours, innocent, of rectum, by C. G. Watson 389

Ulcer of duodenum, the non-surgical treatment of, by G. Herschell 273

Vaccination, value of, by F. M. Sandwith . 346
Vaccine therapy, discussion on . . . 225
Vaccines, use of, in septic and inflammatory conditions 328

Wall, C., foreign bodies in the air-passages . 161
Wallis, F. C., on rectal fistula . . . 369
„ on sigmoiditis and proctitis . 241
„ cases of tubercular disease . 81
Waring, H. J., endosteal sarcoma of tibia . 17
„ carcinoma of rectum treated by abdomino-perinæal method . 193
Watson, C. Gordon, innocent tumours of rectum 389
West, S., clinical lectures on the heart 97, 118, 137
White, W. Hale, on a case of syphilis . . 145
Williams, Leonard, pituitary extract and some of its effects 92
Williams, Tom A., aphasia and other disorders of speech: diagnosis and treatment . . 268

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22474